The New England
Journal of Medicine

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VOLUME 225 JULY 3, 1941 NUMBER 1

MENTAL-HYGIENE CLINICS

SALOMON GAGNON, M.D.*
BOSTON

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MENTAL illness and its related problems are matters for public concern. The community is very sensitive to matters of public health, but generally its interest does not include mental disorders, the greatest and commonest of all menaces. Epidemics of typhoid fever, scarlet fever, diphtheria, infantile paralysis and encephalitis promptly arouse the people and the public authorities to action for preventive and remedial steps, but mental illness,[1] which takes nearly as great a yearly toll in morbidity as all the above combined (72 per cent),[2] does not elicit more than casual notice. The lay public is not alone in this attitude of laissez faire. Professional men and women, including many physicians, do not appear greatly alarmed and, one can safely say, are not cognizant of the magnitude of this menace. Nevertheless it is responsible for the hospitalization of thousands of people each year in this country, and costs much in the way of grief, unhappiness and money. It is time that the people realized that this menace of mental illness is their problem. Much has already been accomplished by a handful of pioneers, but more widespread interest and action are necessary.

In this paper I shall not go into the history of the development of the methods applied to solve this problem, but shall emphasize one procedure that appears to offer great promise. It is generally agreed that there are three principal ways of attacking mental illness: hospitalization of the mental patient after full development of the illness and marshaling, in the hospital centers, the modern methods of care and treatment; prevention by reaching the maladjusted and the predisposed before the illness has reached the stage requiring specialized care and treatment in a mental institution; eradicating these disorders by prophylaxis or specific methods based on increasing the opportunities for research and the discovery of the etiologic factors.

*Chief executive officer, Boston Psychopathic Hospital; instructor in psychiatry, Tufts College Medical School.

Hospitalization is necessary for most of the well-established psychoses, and treatment is carried out in a satisfactory manner in most institutions; this alone is inadequate, however, as witnessed by the marked overcrowding of all mental hospitals.[3]

Should more hospitals be constructed, or should more buildings be added to existing ones? A hospital plant involves a minimum initial outlay of millions, $5,000,000 being about the average figure for a 2000-bed hospital. The annual maintenance cost of such a hospital is approximately $800,000. This is a tremendous burden on the taxpaying public, and an attempt should be made to find less expensive methods of controlling mental disease. It appears desirable, therefore, to slacken the pace of the building program, at least from an economical point of view.

The other two methods mentioned above offer much promise and involve only a small fraction of the cost of constructing and maintaining additional buildings. The expenditure for research in Massachusetts, as well as in other states, is very difficult to determine accurately, but it is most certainly a very small proportion of the total expenditures for mental illness. Research facilities should be greatly increased at strategic locations in hospitals that offer the best opportunities. All hospitals, however, should be given sufficient stimulus to make a contribution.

The purpose of this paper is to stress the value of the so-called "mental-hygiene clinics," including child-guidance and habit clinics. Much has already been done by the establishment of child-guidance and habit clinics throughout the country, particularly in Massachusetts. More adequate co-ordination of the clinic work should be fruitful of results. The significance of the early detection and treatment of the maladjusted cannot be overemphasized, but it seems equally important to expand clinical facilities to include those people beyond the age of fourteen years at the period of their lives when the first obvious manifestations

of mental disease become apparent. There is no definite evidence to prove that the behavior patterns as seen in the child-guidance and habit clinics lead to mental illness or are the prodromal symptoms.

The adult mental-hygiene clinic appears to offer the most practical value. I was connected with such a clinic sponsored by the Danvers State Hospital and operated at the Lynn General Hospital,

TABLE 1. *Types of Cases.*

DIAGNOSIS	NO. OF CASES	
Psychoneurosis		127 (50%)
Neurasthenia	49	
Psychasthenia	31	
Anxiety state	15	
Hysteria	12	
Hypochondriasis	8	
Mixed	6	
Reactive depression	3	
Psychasthenia, with pathologic sexuality	2	
Neurasthenia, with mental defectiveness	1	
Mental deficiency		14 (5%)
Mental deficiency	9	
Mental deficiency, with psychotic episodes	3	
Borderline intelligence, with psychotic episodes	1	
Borderline intelligence, with neurotic traits	1	
Manic-depressive psychosis		11 (5%)
Depressed	10	
Manic	1	
Situational reaction		10 (4%)
Neurotic adolescent		9 (4%)
Epilepsy		8 (3%)
Epilepsy	5	
Symptomatic epilepsy	2	
Epilepsy, with psychosis	1	
Dementia praecox		7 (3%)
Simple	2	
Catatonic	3	
Hebephrenic	2	
Syphilis		5 (2%)
Cerebrospinal syphilis	2	
Juvenile paresis	1	
General paresis	1	
? central-nervous-system syphilis (? epilepsy)	1	
Psychopathic personality		5 (2%)
Psychosis, with cerebral arteriosclerosis	4	
Arteriosclerosis	4	
Mild neurotic tendencies	4	
Epidemic encephalitis (parkinsonism)	3	
Arteriosclerosis, with neurotic symptoms	3	
Senile changes	3	
Primary behavior problem	3	
Chronic behavior problem in adolescence	3	
Miscellaneous	24	
Unclassified	8	
Total	255	

Lynn, Massachusetts. The clinic was held one-half day each week and was conducted by one psychiatrist and one psychiatric social worker. The services of a psychologist were obtained when needed. The appended statistics were compiled to point out the types of cases seen at the clinic and the probable factors involved, and to promote pertinent comments and constructive thoughts.

The total number of patients treated from 1929 to 1934, inclusive, was 255. The diagnosis most frequently made, as shown in Table 1, was psychoneurosis, 127 cases, or 50 per cent of the total, being so diagnosed. Fourteen cases, the next largest group, were diagnosed as mental deficiency, and 11 as manic-depressive psychosis. The diagnosis of situational reaction was made in 10 cases, and that of neurotic adolescent in 9.

It appears, then, that the so-called "functional" or psychogenic conditions comprise the bulk of the cases treated. It is well to note, however, that neurotic symptoms may sometimes be the early or incipient stages of more malignant conditions, such as dementia praecox. These cases, if properly handled, should not progress to the more malignant conditions. There is no way to determine whether or not this will be true of the series reported here, but thus far no case with neurotic symptoms has progressed to either dementia praecox or any other malignant condition.

Forty-seven definitely psychotic patients were also seen at the clinic, including 11 diagnosed as manic-depressive psychosis, 7 as dementia praecox and 5 as central-nervous-system syphilis. Of these psychotic patients only 2 were committed on clinical recommendation or otherwise. Commitment was recommended in another case diagnosed as manic-depressive psychosis, depressed type, because of the severity of the symptoms and the inadequacy of the supervision. This recommendation was not followed, and the patient finally committed suicide. Therefore, of 47 psychotic patients, 44 could be satisfactorily handled at home under clinical supervision and treatment. The majority of these patients would, without question, have been committed if clinical services had not been available. The cost of the hospitalization of this group of 44 patients represents thousands of dollars. It is estimated that the average weekly per capita cost in all the state institutions is approximately $8.00.[4] The average hospital residence of a mental patient is roughly four years. Therefore, on the basis of these figures, the hospitalization of a patient for four years would cost $1664, and the hospitalization of 44 patients on the same basis would amount to a total of $73,216. This apparently represents the saving effected by a clinic very limited in its scope and personnel.

TABLE 2. *Etiologic Factors.*

FACTOR	NO. OF CASES
Economic stress and employment conditions	121
Marital and sexual difficulties	118
Childhood conflicts	116
Physical conditions	84
Social adaptation	26
Hereditary factors	18
Alcoholism	12
Psychic trauma	11
Low degree of intelligence	10
Personality difficulties	8
Religious factors	4

In considering the factors involved (Table 2),

it is noteworthy that only 18 cases, or about 7 per cent, showed heredity factors that could be uncovered by searching social-service investigation. Environmental factors were found to exist in the majority of cases. Economic stress or difficulty in employment was present in 121 cases, or 47 per cent of the total. The economic factor was not the only factor in most cases, and it is difficult to evaluate its relative importance. Physical condition or disease is another significant factor. It was found to be present in 84 patients. Again, its influence on the patient's mental condition is difficult to evaluate. Childhood conflicts of various types were present in 116 cases. These conflicts originated from an oversolicitous mother in 21 patients, unwholesome home in 20, friction between parents in 12, broken home in 11, and a neurotic mother in 11. It may be significant that in only one patient were hardships of childhood considered important. Marital and sexual difficulties were present in 118 cases. Maladjustments of this type are undoubtedly bound up with profound personality difficulties or defects that do not permit adult, mature adaptation. The enumeration of the factors within the home and involving intimate, interpersonal relations looms large in the production of maladjustment, neurotic manifestations and other abnormal mental reactions.

Some emphasis might be placed on the importance of the economic situation in the family circle. Economic stress interferes with the quality of the life of most individuals and of the family group. There is an uncertainty concerning the opportunity of securing the necessities and the niceties of life, and this involves their fundamental, instinctive demands. It impinges daily in a very definite way on the fullness of the life of the individual and interferes with his relations with his fellow men. During a century of industrialism, more emphasis has been placed on the importance of acquisition, and this, along with blatant exhibitions of wealth, has created in the minds of the less fortunate a feeling of envy, dissatisfaction and insecurity. Wealth and economic independence acquired for their own sake, or for the power they endow, are surely not insurance against mental deviations, but they do permit a better and more healthful mode of living. Money, in another way of thinking, also permits indispensable initiatives and the expansion of individual activity. Innumerable frustrations can rightfully be laid at the door of economic inferiority. Gone are the times when people were satisfied with little and content to live submissively with the simple satisfactions that became their heritage. Their sense of relative values has become profoundly modified.

Summary

Those responsible for the care of the mentally ill have been active in promoting the idea of early treatment of incipient and acutely ill patients to prevent much of the accumulation of chronic cases that require costly and prolonged hospitalization.

Specialized clinics of the type discussed in this paper, and institutions dealing primarily with acute material, such as the hospitals in the psychopathic system, appear to be worthy of considerable expansion.

A statistical survey is made of a mental-hygiene clinic dealing with the diagnostic groups treated and the probable factors involved.

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SO-CALLED "MESENTERICOPARIETAL HERNIA"

Report of a Case

HENRY W. HUDSON, JR., M.D.*

BROOKLINE, MASSACHUSETTS

THE following case of imprisonment of the small intestine, owing to a congenital defect in the mesocolon, is reported because of the rarity of the condition.

CASE REPORT

F. R., an 8-year-old schoolboy, entered the Peterboro, New Hampshire, Hospital on August 1, 1939, complaining of severe abdominal pain. The family and past histories were noncontributory except for an indefinite history, extending back for more than a year, of poorly localized attacks of abdominal pain lasting from 1 to 3 hours and unaccompanied by vomiting, fever or evidence of infection.

During the morning of the day before admission, the patient rode horseback and appeared to be well. He refused luncheon and later refused his supper, but made no complaint until the night preceding admission, during which his sleep was restless and he complained of abdominal pain. The boy stated that he had had a stool during the afternoon before admission, but this statement could not be corroborated. By morning he was complaining bitterly of abdominal pain, which he referred to the midabdomen and to the right side. He did not vomit or complain of nausea. On admission the mouth temperature was 98.6°F., the pulse rate 70, and the white-cell count 4000 with 53 per cent polymorphonuclears. The local physical examination was made with difficulty, but his physician, Dr. Donald Clark, found tenderness in the right lower quadrant without other positive physical findings in the abdomen. Rectal examination revealed a large amount of inspissated feces and tenderness more marked on the right side.

Two enemas were administered and were returned with formed fecal masses. Pain appeared to be aggravated by the enemas, and the patient spent the morning in a knee-chest position, which he assumed because of pain. One examination of the urine was negative. At noon the white-cell count had risen to 12,000, and the polymorphonuclears had increased to 90 per cent. There had been no vomiting and no apparent nausea.

Shortly after 1 p.m., I examined the patient in company with Dr. Clark. The child was in bed in the knee-chest position, which could be altered only after great protest. He would consent to be on his left side with the lower extremities flexed on the abdomen, and with great difficulty he was persuaded momentarily to be recumbent with the legs extended. The mouth temperature was then 99°F., the pulse 64. The facies were expressive of pain, the skin was cool and clammy, but he did not appear to be in deep shock. The general physical examination was not remarkable. The abdomen was not distended, and there was no intestinal patterning or visible peristalsis. There was no muscle spasm, but there was excruciating tenderness throughout the right side of the abdomen, with no recognized point of maximum tenderness. Palpation of the left side of the abdomen caused no pain. No masses were felt. Rectal examination was negative. During examination the child appeared nauseated but did not vomit.

Laparotomy was performed at 2 p.m. without a preoperative diagnosis having been made.

Under avertin and ether anesthesia, an incision, which was eventually extended from the right costal margin to the pubis, was made and the peritoneum incised without incident. The omentum presented, and with it the large intestine, identified as the transverse and left colon. The stomach, liver and gall bladder were normal in appearance and position. A large, thin-walled, transparent hemispherical peritoneal sac occupied the greater part of the abdominal cavity. Through the sac, coils of intestine were visible. The sac was incised, exposing the small intestine, appendix and cecum. The exposed small intestine was slightly cyanotic, and there was a volvulus of 180° in a clockwise direction. This was reduced, with improvement in the color of the bowel. With the volvulus reduced, the entire small intestine lay anterior to the transverse colon and toward the right side of the abdomen. Further exploration, with the transverse colon and omentum elevated, revealed a defect in the mesocolon adjacent to the last part of the duodenum and ligament of Treitz. Through this defect, the small intestine appeared to have passed progressively, beginning with the jejunum and continuing until the appendix, cecum and ascending colon had also passed through the defect. By exerting traction, — first on the right colon, then on the cecum, then on the ileum and finally on the jejunum, — the small intestine was withdrawn until it came to occupy its normal position below and behind the transverse colon. With this accomplished, the color of the bowel was good, and it was possible to place the intestinal tract in its normal position. The cecum and ascending colon were unduly mobile, and the ascending mesocolon was represented by a peritoneal reflection from the right iliolumbar fossa.

These several manipulations were not accomplished without the expenditure of a good deal of time and handling of the bowel. It therefore seemed unwise to prolong the operation by seeking to determine the distribution of the blood supply to the bowel. The abdominal wound was closed without drainage, intravenous glucose was administered, and the child was returned to bed in good condition without evidence of shock and with a pulse of 110 and a systolic blood pressure of 104.

Convalescence was uneventful, and the patient was discharged from the hospital 12 days after operation.

By September 28, 1939, he had gained 6 pounds, had returned to school, and there had been no complaints referable to the abdomen. The report of an x-ray examination by Dr. E. C. Vogt on this date was as follows:

> A flat abdominal film shows the kidneys to be normal in size and position. There are no abnormal masses or calcification.
>
> The barium enema fills the colon easily and well. The

*Assistant in surgery, Harvard Medical School; associate surgeon, Children's Hospital.

colon is normal in contour, and it is in proper position. The cecum is rather low. There is no obstruction. Examination after evacuation shows complete emptying of the colon.

In a gastrointestinal series, the esophagus appears normal. The stomach is medium in size and position and normal in contour and empties freely. The duodenum appears normal. Films taken at intervals up to 45 minutes very well delineate the small intestine, which is normal in appearance. There are no obstructions or constrictions, and the position is normal. The mucosal pattern is well shown, and entirely normal. The head of the barium column at 45 minutes is at the hepatic flexure.

Impression: normal gastrointestinal tract.

On June 12, 1940, the patient entered the Children's Hospital (Boston). In the fall of 1939 he had complained of transient abdominal pain on several occasions, but the pain was neither severe nor localized. He had been considered to have a good deal of "gas." Otherwise he had been well, and his schooling had not been interrupted. He was admitted to the hospital for abdominal exploration and closure of the aperture noted at the former operation.

On June 14, an operation was performed. Under avertin and ether anesthesia a transverse upper abdominal incision was made, with division of both rectus muscles and the external oblique aponeurosis, and retraction of the internal oblique muscle. This incision gave a very good exposure. The first abnormality encountered was a tense, stringlike adhesion about 15 cm. long, running from the ileum to the transverse colon. This was ligated and divided. The inferior edge of the omentum presented as two layers that were not fused, so that the omental bursa was open. The transverse colon and the omentum were displaced upward, and the small bowel was packed away, exposing the root of the mesentery and the superior mesenteric vessels. Just below these vessels and directed upward to the right was a slitlike aperture approximately 2 cm. in height. Through this aperture a No. 20 rubber catheter was easily passed for a distance of 15 to 20 cm.; it went to the right, and presented behind and lateral to the upper portion of the ascending colon just below the hepatic flexure. After this demonstration, the catheter was withdrawn, and the edges of the aperture described were roughened with gauze and then approximated with four interrupted, fine-silk sutures. Another adhesion running from a loop of ileum to the inferior end of the previous abdominal scar was divided. This adhesion was about 2 cm. in width and was relatively vascular. The cecum was mobile and easily delivered, and with it an extremely long but otherwise normal appendix. The mesoappendix was ligated and divided. The appendix was crushed at its base, ligated and removed with the cautery. The intestine was placed in normal position. The free edge of the omentum, which presented in two layers, was approximated by means of a series of interrupted sutures resulting in the closure of the omental bursa. The abdominal wound was closed in layers, without drainage. Interrupted black-silk sutures were used throughout, except for ligation of the appendiceal stump and for the peritoneal closure, the latter being approximated by means of a continuous catgut suture. The patient was returned to bed in good condition.

Convalescence was entirely uneventful, and he was discharged on June 25.

He was seen on November 21, when he was in excellent health, had gained 8 pounds in the preceding 5 months and was actively engaged in schoolboy athletics. Both wounds were solidly healed, and there had been no complaints.

Discussion

The anatomic abnormality in this patient is explained embryologically by "the imprisonment of the small intestine beneath the mesentery of the developing colon" (Longacre[1]), and is not a hernia in the sense of the term that implies protrusion or evagination of the intestine from the celomic cavity. During the third stage of return of the intestinal tube from its temporary extrusion into the yolk sac — in the fifth to eleventh weeks of embryonic life — the mesocolon (in this case, the transverse mesocolon) may fail in part to fuse with the posteroparietal peritoneum. This affords opportunity for the midgut to be covered by or imprisoned behind the mesocolon, and it is the mesocolon that presents the appearance of a covering or enveloping peritoneal sac.

Excellent clinical and anatomic descriptions are available in the recent American literature, and it appears unnecessary to reproduce them, although it is desirable to refer to the sources. Historically, Neubauer[2] appears to have described the condition first; Treitz's[3] description in 1857 is classic, and the several allied conditions are often referred to as "Treitz hernias," since he believed that the intestine deepened and protruded into one of the several peritoneal fossas about the duodenum. Moynihan[4] published a monograph on the subject in 1906. Bender[5] suggested a developmental, as opposed to an acquired, mechanism, and Eisler and Fisher[6] and Bender[5] introduced the term "mesentericoparietal hernia" to replace Treitz and paraduodenal hernia (right and left) and to distinguish these anomalies from internal hernias, many of which bear no anatomic relation to the condition under discussion.

Two excellent papers, based on anatomic dissections, — one by Longacre[1] and one by Callander, Rusk and Nemir,[7] — discuss the type of anomaly presented in the reported case. Both are fully and beautifully illustrated and serve to make clear the embryologic basis for a puzzling clinical picture. Longacre's bibliography is particularly complete. These papers, published respectively in 1934 and 1935, have been followed by numerous articles up to and including June, 1940.

Bryan[8] described the case of a nineteen-year-old girl treated successfully, and stated his to be the forty-third reported case of right paraduodenal hernia and the fifteenth patient to recover.

Emenhiser and Pankratz[9] reported on necropsy observations in a case of left paraduodenal hernia, and Averbach[10] noted the successful treatment of a fourteen-year-old boy presenting findings very

similar to those of the patient reported in this paper.

Paul and Hill[11] commented on the autopsy findings in a three-month-old female and discussed the embryology from a viewpoint slightly different from those of the authors referred to above.

Halpert,[12, 13] in 1938 and 1939, reported postmortem studies of a thirty-five-year-old woman with a right paraduodenal hernia and of a fifty-five-year-old man with a left-sided hernia, respectively. He states in the 1938 paper that he presents the forty-ninth confirmed case of right paraduodenal hernia.

Snyder[14] presented another case, not included by Halpert, of successful surgical reduction, but in this case, death followed forty-eight hours after operation. The patient was a man of fifty-one with extensive pulmonary tuberculosis.

Johns[15] reported a left transmesocolic hernia and emphasized the importance of a significant symptom — the pain, however severe when standing, is relieved when the patient lies down. His patient was a man of twenty-eight years.

Hansmann and Morton[16] presented the case of a fifty-eight-year-old cretin whose hernia occurred through the external leaf of the mesentery of the ascending colon, and reproduced the roentgenograms, with and without barium, after the cecum had become obstructed. These authors review the subject and give a bibliography but include hernias that were sequelae of abdominal operations involving the several mesenteries. They make the valuable observation, based on statistics, "that in most instances the stoma of intra-abdominal hernia will be found by lifting up the transverse mesocolon and exploring the paraduodenal region."

Developmentally the condition described is intimately related to and may present symptomatology similar to the extrinsic obstructions stressed by Ladd.[17–21] In these obstructions, owing to incomplete rotation of the colon and defective mesenteric development, there may be a volvulus of the small intestine and compression of the duodenum by the overlying and abnormally placed cecum and ascending colon. Haymond and Dragstedt[22] have also described anomalies of intestinal rotation.

For references to imprisonment of segments of the small intestine in apertures in the paracecal ascending colon, there are articles by Chamberlain[23] and by Cullen.[24] A third case (C. H. No. 223661), not reported but successfully treated by resection of part of the ileum, was operated on by Dr. T. W. Botsford at the Children's Hospital in August, 1939.

The patient reported here differs from others previously mentioned because the imprisonment was by the transverse mesocolon rather than by the ascending or descending mesocolon, because volvulus of the small intestine was present, and because the terminal ileum, cecum and part of the ascending colon were imprisoned, as well as the jejunum and upper ileum, which have been reported before. These are not discrepancies, however, since in addition to either the ascending or the descending mesocolon, the transverse colon may fail to fuse to the posterior peritoneum. Furthermore, all the imprisoned bowel was, embryologically speaking, part of the midgut — that is, the part supplied by the superior mesenteric artery. The presence of volvulus of the small intestine is quite uniformly found in the anomalies described by Ladd.[17–21]

Longacre[1] summarized the evidence for the belief that such cases are congenital anomalies by pointing out the size and extent of the so-called "sacs," the absence of a definite neck, the absence of entering and departing loops, the absence of omentum in the sacs, and peculiarities in the blood supply that cannot be explained as acquired. In the present case the exigencies of operation prevented observation of anomalies of blood supply, but to Longacre's evidence may be added the imprisonment of essentially all segments of the intestine arising from the midgut, the defective mesenteric attachments and the volvulus, which also occurred in the undoubtedly congenital anomalies described by Ladd.

Clinically the condition, although rare, bears an intimate relation to other forms of intestinal malrotation and reverse rotation that may produce obstructive phenomena in the newborn or later in life. It may also be the cause of symptoms similar to those produced by internal herniations into apertures in the broad ligament and omentum, and by incarceration of knuckles of bowel into the several pericecal and paraduodenal fossas.

It appears from the data reported that cases of this type are congenital anomalies of development of the intestinal tract, that they may exist without producing functional disturbance, that they may be responsible for long-continued but vague symptoms of disturbed intestinal function, and that they may produce acute obstruction phenomena, with varying degrees of circulatory impairment of the bowel. Andrews[25] states that the treatment of strangulation appears to be almost hopeless, reduction being nearly 100 per cent unsuccessful. He adds that unless strangulation takes place, the bowels are able to function normally.

About 50 cases of so-called "herniation" into the right mesocolon have been reported, about 150 into the left, and 2 into the paracecal segment of

the right colon; the present case is considered to be imprisonment by the transverse mesocolon.

Correct preoperative diagnosis is seldom possible, a total of 5 having been reported by Abrazon, von Haberer, Vautrin and Slandenmeyer (Longacre[1]). The use of x-ray examination has been suggested, and Snyder's[14] patient did present an appearance suggestive of incarceration of many loops of intestine. The existence of a tympanitic circumscribed mass has been described, but apparently has not been found in most cases. The usual symptoms are those of intestinal obstruction with varying degrees of shock. Pain appears to be the most constant symptom, and together with abdominal tenderness, may be the indication for operation. In this connection, one can do no better than to quote with Chamberlain[23] the statement of Masson and Atkinson[26]: "Time spent in differential diagnosis is ill spent. The decision to be made is whether or not the condition is surgical. The chagrin of the surgeon who operates and fails to find a surgical condition is nothing to that of the surgeon who does not operate in time."

Successful treatment postulates early operation before serious circulatory disturbances have developed, and a reasonably accurate knowledge of embryologic development so that a rational plan for relieving the intestinal obstruction and the vascular obstruction may be employed.

Summary

A case of imprisonment of the segments of the intestine arising from the midgut by a congenital anomaly of the transverse mesocolon is presented, it is believed, for the first time; the successful surgical management of the patient is described.

The embryologic explanation for the anomaly is briefly described, and references are given to the descriptions of the related anomalies of so-called "herniations" into or imprisonment by the ascending and descending mesocolons. The clinical and anatomic relations to other forms of faulty rotation and attachment of the colon are discussed. Additional evidence for the developmental basis for such anomalies is added — namely, the imprisonment of essentially all the intestine arising from the midgut, the defective mesenteric attachments and the coexistent volvulus of the small intestine. The designation of "hernia" is retained, since it is a useful and convenient term, although the anomaly is not a hernia in the usual sense.

1101 Beacon Street

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GALLSTONES IN PATIENTS UNDER THIRTY YEARS OF AGE

CARL BEARSE, M.D.*

BOSTON

ALTHOUGH approximately 10 per cent of the population have gallstones,[1] the prevalence of this condition in patients beyond forty years of age has obscured its relative frequency in young persons. An analysis of 300 consecutive cases of operation for gall-bladder disease, including noncalculous cholecystitis,[2] disclosed that 21 per cent of the patients were under thirty years of age. It has been suggested[3] that if routine cholecystography were carried out in young persons, biliary-tract disease would be found even oftener.

In the present study of 232 unselected patients operated on for gallstones, 48, or 20 per cent, were under thirty years of age. There were 3 males and 45 females. The males were in their third decade, as were 41 of the females. The remaining 4 females were operated on before their twentieth year, the youngest being fourteen years of age.

Previous acute infections, including the exanthematous diseases, have been suggested as etiologic factors in the formation of gallstones in young persons. Twenty-seven patients (56 per cent) gave a history of one or more of these infections. Measles was the commonest, and whooping cough and scarlet fever were next in frequency. There were several cases of typhoid fever, chicken pox and mumps, 1 of smallpox and malaria, and 1 of influenza.

More than 50 per cent of all patients with gallstones have been reported to be obese,[4-6] but only 21 per cent of these young patients were overweight; 11 per cent were thin, and 68 per cent were of normal weight.

The symptoms were no different from those in other age groups. Although all these patients had pain to some degree, only 18, or 38 per cent, at times required morphine by hypodermic injection. In comparison with reports for older patients, nausea and vomiting (in 40 per cent) were approximately as frequent, but eructations (in 18 per cent) were about one fourth as common. In 6 patients (13 per cent) jaundice was present at the time of operation or had been observed during previous attacks.

The duration of symptoms varied from one day to seven and a half years (Table 1). The average was one year and eleven months, approximately four years less than the average for patients of all ages.[7, 8]

Approximately 70 to 90 per cent of women operated on for gallstones have symptoms in relation to pregnancy[9]; of the 36 married women in this series, 18 (50 per cent) had attacks during the gravid state. The frequency of attacks during and shortly after gestation has been attributed to hypercholesterolemia, impairment of gall-bladder function and constipation. One patient, aged twenty-eight years, had attacks of pain so severe that the gall bladder was removed during the fourth month of pregnancy. She had an uneventful convalescence and no further symptoms during or subsequent to her pregnancy and confinement.

TABLE 1. *Duration of Symptoms.*

DURATION	NO. OF CASES
Less than 1 yr.	15
1 to 2 yr.	11
2 to 5 yr.	17
Over 5 yr.	4
Unknown	1
Total	48

Six patients (13 per cent) had acute cholecystitis in addition to cholelithiasis. Four had symptoms for two months or less; the remaining 2, one and a half and seven and a half years, respectively. The gall bladders were viable, although in 1 case the mucosa was gangrenous. The fact that the symptoms did not exceed eight weeks in 4 of these patients indicates that acute cholecystitis in young persons may occur early during the course of cholelithiasis.

The common duct was opened and explored in 8 cases; stones were found in 3, an incidence of only 6 per cent for the series. These patients, whose ages were between twenty-four and twenty-seven years, had symptoms from four to seven years; none were jaundiced at the time of operation, and only 1 gave a history of jaundice. As in older patients, the longer the duration of symptoms, the greater the incidence of this complication.[10] For patients of unselected ages, 16 to 21 per cent are estimated to have choledocholithiasis,[11, 12] with jaundice in 60 to 91 per cent.[12, 13]

Three patients (6 per cent), who were twenty-six, twenty-seven and twenty-nine years of age, respectively, had acute pancreatitis in addition to

*Associate visiting surgeon, Beth Israel Hospital.

cholelithiasis. One had edema of the pancreas, and the other 2 had hemorrhagic pancreatitis with fat necrosis. One was operated on within twenty-four hours of the onset of the first gallstone attack, and the others had biliary symptoms for one and three years, respectively. It is therefore obvious that young patients with gallstones can have acute pancreatitis as an early complication.

There were no deaths in the series. The gall bladder was removed in each case, and, as already mentioned, the common duct was opened and explored in 8. Thirty-eight patients (79 per cent) also had an appendectomy, including 7 who had additional surgery.

Forty-six patients (96 per cent) had an uneventful convalescence and were discharged from the hospital within two weeks of operation. One of the remaining patients was in the hospital thirty-five days because of a phlebitis, first of one leg and then of the other. The second patient, whose common duct was drained, had a nonvirulent gas-bacillus infection necessitating twenty-one days of hospitalization.

It has been possible to follow 38 patients (79 per cent) for a period of one to fourteen years. Only 2 have thus far complained of subsequent distress. One patient had epigastric discomfort two years postoperatively; roentgen-ray examination revealed a duodenal ulcer. The other, after being free from symptoms for four years, had an attack of epigastric pain that persisted for several days. Careful investigation, including a gastrointestinal roentgen-ray examination, was negative. Whether the recurrence of pain in this patient was due to an overlooked common-duct stone or to an extrabiliary condition has not yet been determined. The 3 patients who had stones removed from the common duct have been free from symptoms for two to four years.

Summary

Of 232 consecutive operations for cholelithiasis, 48 (20 per cent) were on patients under thirty years of age. The average duration of symptoms was one year and eleven months.

In addition to cholelithiasis, 6 patients (13 per cent) had acute cholecystitis, 3 (6 per cent) had acute pancreatitis, and 3 (6 per cent) had stones in the common duct.

The gall bladder was removed in each case. There were no deaths, and postoperative complications occurred in only 2 patients.

Of the 38 patients (79 per cent) who have been followed for more than one year only 1 developed symptoms that might have been related to the biliary tract, and these occurred four years after operation. There has been no recurrence of symptoms in those who had common-duct stones.

483 Beacon Street

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MASSACHUSETTS MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND SIXTIETH ANNIVERSARY

THE one hundred and sixtieth anniversary of the Massachusetts Medical Society was observed in Boston on Wednesday and Thursday, May 21 and 22, 1941, at The Copley-Plaza.

WEDNESDAY, MAY 21

A general clinical meeting was held in the Sheraton Room in the forenoon, under the chairmanship of Dr. Bennett F. Avery. The attendance was approximately 425.

The supervising censors met in Parlor A at 10:00 a.m., and the Council convened in the Swiss Room at 10:30 a.m., the Cotting Luncheon being served at 1:00 p.m.

During the noon hour, the sections met for a series of round-table discussions and luncheons, at which the new officers were elected. The Section of Medicine met at the University Club, with an attendance of 148; the Section of Obstetrics and Gynecology at the University Club, with an attendance of 77; the Section of Surgery in the State Salon of The Copley-Plaza, with an attendance of 85; the Section of Pediatrics at the Hotel Lenox, with an attendance of 56; the Section of Dermatology and Syphilology at the Hotel Lenox, with an attendance of 53; the Section of Radiology and Physiotherapy at the Hotel Lenox, with an attendance of 13; and the Massachusetts members of the New England Society of Anesthesiology at The Copley-Plaza, with an attendance of 35.

In the afternoon, a general clinical meeting was held in the Sheraton Room, Dr. C. Sidney Burwell presiding. The attendance was around 450.

That evening, the annual dinner was held in the Sheraton Room, 281 members and guests being present. The president, Dr. Walter G. Phippen, introduced the delegates from the medical societies of the other New England states, as follows:

MAINE: Dr. Theodore E. Hardy, Waterville

NEW HAMPSHIRE: Dr. Clinton R. Mullins, Concord
Dr. Leslie B. Copenhaver, North Woodstock

VERMONT: Dr. Michael F. Cerasoli, Barre

RHODE ISLAND: Dr. Charles A. McDonald, Providence
Dr. Joseph C. O'Connell, Providence

CONNECTICUT: Dr. Charles H. Turkington, Litchfield
Dr. F. Erwin Tracy, Middletown

After short speeches by Drs. Nathan B. Van Etten and Frank H. Lahey, president and president-elect, respectively, of the American Medical Association, the attending fellows adjourned to the Swiss Room, where Dr. E. W. Alton Ochsner, of New Orleans, delivered the Shattuck Lecture, his subject being "Thrombosis and Thrombophlebitis." (This lecture will appear in the July 31 issue of the *Journal*.)

THURSDAY, MAY 22

A symposium on hormones was held during the early forenoon, under the chairmanship of Dr. Charles E. Mongan; the attendance was approximately 425.

ANNUAL MEETING

The one hundred and sixtieth annual meeting was called to order by the president, Dr. Walter G. Phippen, in the Sheraton Room at 11:00 a.m. Approximately 400 were in attendance. The minutes of the previous annual meeting were approved as published in the July 4, 1940, issue of the *New England Journal of Medicine*. The Secretary *pro tempore* then presented the changes in membership, as follows:

Membership reported May 22, 1940			5542
Losses			
Deaths	104		
Resignations	17		
Deprivations	49		
		170	
Gains			
New fellows	238		
Readmitted by censors	1		
Restored by Council	28		
		267	
Net gain			97
Membership reported May 22, 1941			5639

Proposed changes in the by-laws, which had previously been approved by the Council and copies of which had been forwarded to all members of the Society, were then considered. Dr. Phippen called attention to the fact that the last

four proposed amendments had been inadvertently omitted from the notice of the annual meeting and that a postcard calling attention to the omission had been sent to every member. The amendments were considered in order, and were accepted without change, except that in Amendment 17 the word "Society" in the next-to-the-last line in the first paragraph was changed to "Council."

The amendments, as accepted, were as follows:

AMENDMENT 1. Amend Chapter I, Section 1, to read:

Applicants for admission to fellowship in the Massachusetts Medical Society are required to satisfy the censors that they are not less than twenty-one years of age; that they are of sound mind and of good moral character; that they possess a good English education; that they have received a diploma from a medical school or college recognized by the Council, or that they have, in each instance, received the approval of the Committee on Medical Education; that they do not practice medicine in a manner contrary to the code of ethics of this society; that they have made application according to the provisions of Chapter V, Section 2; that they have paid the examination fee of three dollars; and they shall appear personally before the censors and satisfy them that the above requirements are fulfilled.

AMENDMENT 2. Amend Chapter I, Section 6, to read:

Fellows shall be assessed annually such a sum as the Council may determine. The fiscal year shall begin on the first day of January. Assessments shall be payable in advance. The first assessment paid by a fellow admitted to the Society following the December examinations shall cover his dues for the succeeding fiscal year and shall be the amount fixed for that year. . . .

AMENDMENT 3. Amend Chapter I, Section 7, to read:

The resignations of fellows whose assessments have been paid in full or remitted, may, on recommendation of the Committee on Membership, be accepted by the Council. Petitions to be allowed to resign should be sent to the treasurer of the general society.

AMENDMENT 4. Omit the last paragraph of Chapter I, Section 8.

AMENDMENT 5. Add to Chapter I the following:

Section 10. Former fellows who desire to be readmitted to the Society shall make application in writing addressed to the Council and sent to the secretary of the Society. Such applications shall be referred for investigation and personal interview to the local boards of membership, which shall report their recommendations to the Committee on Membership. The committee shall render the final decision as to whether to recommend to the Council the readmission of former fellows. The Council shall have the power to readmit former fellows so recommended. Boards having under consideration the applications of fellows whose resignations have been requested by the Committee on Ethics and Discipline, or who have been deprived of fellowship under the terms of Section 8, Clause (*c*), shall consult with the Committee on Ethics and Discipline before reporting their recommendations.

AMENDMENT 6. Add to Chapter III, Section 5, the following:

The councilors of each district society shall meet at or as soon as possible after the annual meeting of the district society in 1941 and elect one of their number to serve as a member of the Executive Committee of the Council, in accordance with Chapter IV, Section 10. Thereafter, in a similar manner, they shall elect a new member from time to time.

AMENDMENT 7. Add to Chapter III, Section 7, the following:

The secretary of each district society at or as soon as possible after the annual meeting of the district society and before the annual meeting of the general society in 1941 shall call together its councilors for the purpose of electing a member of the Executive Committee of the Council and shall send the name of the member chosen to the secretary of the general society. He shall see that a new member is chosen in a similar manner to fill a vacancy as it occurs, in accordance with Chapter IV, Section 10.

AMENDMENT 8. Add to Chapter IV the following:

Section 10. The Executive Committee shall consist of the President, President-Elect, Vice-President, Secretary and Treasurer *ex officiis*, and a councilor from each district medical society chosen in accordance with Chapter III, Section 5.

The councilors of each district society shall meet at or as soon as possible after its annual meeting in 1941, as specified in Chapter III, Section 5, and in a similar manner at stated intervals as specified below, to choose one of their number to serve as a member of the Executive Committee of the Council.

A member of the Executive Committee shall serve for three years and shall not be eligible for re-election before the expiration of three years following the conclusion of his term of office; except that in the first year of operation six members shall be selected by lot to serve one year, six members shall be selected by lot to serve two years and six members shall be selected by lot to serve three years. Thereafter, six members shall be elected every year to succeed in office those whose terms are about to expire.

The Executive Committee shall meet at the call of the President at least once in each interval between Council meetings and may meet more often at the pleasure of the President. It shall assist the President in preparing for the consideration of the Council matters calling for action by the Council at its next meeting. It shall authorize action by the officers of the Society when circumstances require it, subject to the approval of the Council. It shall perform such other duties as the Council may require.

An executive secretary may be chosen by the Executive Committee upon nomination by the President.

Upon request, members of the Executive Committee shall be paid the amount of their traveling expenses from the funds of the Society.

AMENDMENT 9. Amend Chapter V, Section 1, to read:

The supervisors, representing the censors of the several district societies, shall constitute a board, which shall meet annually on the day appointed for the annual meeting of the Council. The board shall elect a chairman, who shall have power to call special meetings. Five supervisors shall constitute a quorum. The secretary or executive secretary of the general society

shall act as secretary of the board. He shall call special meetings at the request of five supervisors. He shall keep a permanent record of the proceedings of the board, and shall provide, at the expense of the Society, papers and forms necessary for conducting examinations of applicants for fellowship. The board at its annual meeting shall adopt a uniform plan for the examination of applicants. The supervisors shall be paid the amount of their traveling expenses from the funds of the Society.

The supervisors shall be chairmen of their respective boards of censors, and shall cause the examinations of applicants to be conducted in strict conformity to the plan adopted by the Board of Supervisors.

The censors of the several district societies shall meet semi-annually for the examination of applicants on the first Thursday in May and on the first Thursday in December.

A fee of three dollars shall be paid to the district secretary by an applicant for fellowship for each examination or re-examination by the censors. This shall be sent promptly to the treasurer of the general society for deposit in the funds of the Society. An applicant shall not be considered as possessing the requisite qualifications for fellowship unless approved by at least three censors. An applicant failing two examinations shall be disqualified from again applying for fellowship until three years have elapsed from the date of the last application.

AMENDMENT 10. Change Chapter V, Section 2, to read:

(*a*) An applicant for fellowship who is a graduate of a medical school or college recognized by the Council shall apply on a form furnished for the purpose to the secretary of the district in which he has legal residence not later than February 15 for the May censors' examination or September 15 for the December censors' examination. At this time the district secretary shall verify the applicant's diploma and shall deliver the application form to the secretary of the general society not later than February 20 or September 20 respectively. An applicant nonresident in Massachusetts shall apply to the secretary of the Suffolk District Medical Society and shall be examined by the censors thereof. Consideration of a late application shall be postponed until before the next succeeding examination. The names of all such applicants, their addresses, medical schools and colleges, dates of graduation and the names and addresses of the various district secretaries shall be published in a list in the first number of the *New England Journal of Medicine* on or after March 5 or October 5. Confidential communications regarding the qualifications of applicants for fellowship shall be requested of the fellows of the Society to be sent to the appropriate district secretary not later than March 15 or October 15.

(*b*) An applicant for fellowship who is a graduate of a foreign medical school or college or a domestic medical school or college not on the list recognized by the Council or of a medical school or college no longer in existence, and who has practiced for a minimum of five years, shall apply for fellowship in like manner with the following exceptions and additions:

At the time of application the applicant must submit the name and address of a fellow of the Society who has agreed to act as his sponsor. The sponsor's duty is to obtain from fellows of the Society who are acquainted with the applicant and his work, confidential written opinions regarding his qualifications for fellowship to be mailed directly to the district secretary not later than March 15 or October 15. The list of these applicants published in the *New England Journal of Medicine* shall also include the names and addresses of the applicants' sponsors.

The president, secretary and supervising censor of the district society, sitting as a local board of membership, shall then gather such further information as is deemed necessary to determine whether an applicant is a capable and conscientious practitioner of medicine and possesses a good professional reputation among his colleagues. Every candidate must be personally interviewed by this board.

The district secretary shall deliver to the chairman of the Committee on Medical Education a complete confidential file of all applications including correspondence, and the written recommendations of the local board with supporting reasons for advocating the acceptance or refusal of each applicant not later than April 1 or November 1. The Committee on Medical Education shall then determine whether or not each candidate shall be approved for examination by the censors, and shall notify the district secretary and each applicant of its decision not later than April 20 or November 20.

AMENDMENT 11. Amend Chapter V, Section 3, to read:

District secretaries, who shall act as secretaries of their respective boards of censors, shall furnish applicants with forms adopted by the Board of Supervisors; they shall keep a complete record of all applicants for fellowship; they shall see that each applicant pays the examination fee; . . .

AMENDMENT 12. Add to Chapter VI, Section 1, the following:

He shall call at least one meeting of the Executive Committee of the Council between Council meetings and may call more meetings if he so desires.

AMENDMENT 13. Change Chapter VI, Section 3, to read:

The Secretary may assign to an executive secretary any or all the duties now to be enumerated, except as specified below:

The Secretary shall attend all meetings of the Society and of the Council, and shall record their respective proceedings in separate record books; and this duty he may not assign.

He shall cause to be engrossed and shall sign the diplomas of new fellows if satisfied that they have met the requirements of Sections 1 and 2 of Chapter I, and shall issue all diplomas and certificates of fellowship. He shall notify individual fellows, in appropriate instances, of votes by the Council granting permission, as the case may be, to retire, to resign, to change district membership, to have dues remitted, or of votes depriving them of the privileges of fellowship; and these duties he may not assign.

He shall act *ex officio* as secretary of all boards of trial; and this duty he may not assign.

He shall have custody of the seal of the Society and of all books, papers, manuscripts, prints and paintings belonging to the Society, except such as are in charge of the Treasurer; and this duty he may not assign.

He shall act *ex officio* as secretary of the Board of Supervisors and of the Committee on Publications and the Committee on Ethics and Discipline, and shall keep the records of each in a separate volume. He shall have custody of all records as thus kept.

He shall issue notices of the meetings of the Council. One month before the annual meeting of the Society, he shall issue to every fellow a program, which shall contain notification of the time and place of the annual meeting, notification of the stated meetings of the Council for the year and the meetings of the boards of censors, and information concerning the payment of assessments and the distribution of publications.

He shall keep a complete list of the fellows of the Society, with their addresses so far as known. He shall transfer fellows from one district to another under the terms of Chapter III, Section 2, and shall report to the Society at its annual meeting the changes in membership of the Society during the year.

He shall conduct the official correspondence of the Society, and shall notify officers, delegates and members of the committees of the Society of their appointments and of their duties.

Under the direction of the Committee on Publications he shall issue at such intervals as may be determined by the Council a directory of officers and fellows of the Society, which shall be furnished upon request to fellows who are not in arrears.

He shall perform such other duties as the Society or the Council may require.

AMENDMENT 14. Change Chapter VI, Section 5, to read:

The Executive Secretary shall hold office at the pleasure of the Executive Committee of the Council.

He shall perform such duties as are assigned to him in Section 4 of this chapter and by the Executive Committee. In general, he shall assist the officers of the Society and such standing and other committees as may request his services, and the Society shall have the first call upon his services.

He shall attend all meetings of the Council and, on request, attend the meetings of the Executive Committee of the Council, but shall not vote in either.

The Executive Secretary need not be a physician.

AMENDMENT 15. Amend Chapter VII, Section 5, to read:

The Committee on Medical Education shall consist of five fellows. It shall consider all matters relating to medical education which may be referred to it by the Council. It shall review the case of every applicant for fellowship who presents a diploma according to the provisions of Chapter V, Section 2(*b*). It shall have the power to approve for examination by the censors such an applicant, and all decisions of the committee thereon shall be final. It shall revise the list of medical schools and colleges recognized by the Council annually for approval by the Council. . . .

AMENDMENT 16. Amend the first paragraph of Chapter IV, Section 1, to read:

The Council shall consist of councilors chosen by the district societies, and the president, ex-presidents, president-elect, vice-president, vice-presidents *ex officiis*, secretary and treasurer of the general society, secretaries of the district societies, and the chairman of each standing committee.

AMENDMENT 17. Amend the first paragraph of Chapter IV, Section 3, to read:

The Council at its annual meeting, on nomination by the Nominating Committee and/or from the floor, shall elect by ballot officers of the Society as follows: president-elect, who shall serve as president-elect until the annual meeting of the Society next ensuing after his election and shall become president on his installation in the course of that meeting, serving thereafter as president until the next following annual meeting and the installation of his successor; a vice-president, secretary and treasurer, all of whom shall assume the duties of office at the close of the annual meeting of the Society and shall hold office until their successors have been duly elected; except only that at the annual meeting of the Council in 1941 there shall be nominated and elected a president to serve for that year.

In the absence of a president-elect, the Council, at its next annual meeting, shall upon nomination by the Nominating Committee and/or from the floor, elect a president. Councilors only shall be eligible to the offices above named. Upon nomination by the Nominating Committee, the Council shall elect by ballot a fellow to deliver an oration at the annual meeting of the Society the following year.

AMENDMENT 18. Change Chapter VI, Section 3, to read:

The President-Elect shall assist the President in the performance of his duties in such a manner as the President may direct and in so doing shall be considered to represent the President.

AMENDMENT 19. Change Sections 3, 4 and 5, Chapter VI to Sections 4, 5 and 7, respectively.

Dr. Leroy E. Parkins moved that a vote of thanks be extended to the Committee of Arrangements for the very excellent program that had been presented; the motion was seconded and carried.

Dr. Phippen then gave his report on the state of the Society:

It is my duty at this time to report on the state of your society. It is a pleasure to say that it continues to grow and prosper. Its activities are expanding. Its influence is beginning to be felt. A larger proportion of members are giving more thought to the affairs of the parent society. The attendance at Council meetings has been very gratifying and denotes, I believe, a more careful selection of councilors by the district societies. Only men who are willing and able to give freely of their time and thought should be elected to these important positions.

The total membership now numbers 5639, an increase of 97 in the past year.

The Society sustained a great loss during the year in the death of its very able secretary, Dr. Alexander S. Begg. Dr. Begg was devoted to the interests of the Massachusetts Medical Society. He knew its history and its traditions. He probably knew more members intimately than any other member. Moreover, he was a keen judge of men, and knew who could be entrusted to do any particular job well. We miss him sadly, but in so doing, rejoice in his acquaintance and do honor to his service.

Our able and devoted treasurer reports again that our finances are as sound as the times will allow. He has continued his policy of placing funds of the Society in bonds, and in most cases only in the best. One result of this is lower interest rate, but with greater security of principal. At present the Society has a large amount of cash awaiting investment, owing mainly to the receipt of 1941 dues; the Treasurer has not been able to invest

all in securities fit, in his opinion, for our funds. Almost all the Society's holdings of bonds are well above the cost prices. The Society, so far in 1941, has received from securities sold or from securities called for early payment a profit of several hundred dollars. No default in interest receivable has occurred this year, other than the issues already in default for several years. Collections of annual dues from resident fellows, to May 1, are greater than in 1940 for the same period. Nonresident dues received are also greater than in 1940. Expenses through April, 1941, have shown increases, but not to an unreasonable degree.

The Treasurer regrets that he feels obliged to carry such a large amount of cash awaiting investment. This idle cash should be earning income for the Society. The Treasurer is seeking, and occasionally finding, what he thinks is a proper medium for our funds.

A goodly number of fellows of the Society have been called to active duty with the United States Army and Navy. Some of these have asked to have their 1941 dues remitted.

Again I congratulate the Society on the wisdom and sagacity of its treasurer, and again I thank the Treasurer in the name of the Society for his conscientious and able service.

The standing committees have continued their efficient work.

It is a pleasure to note that the Committee on Ethics and Discipline has had rather less troublesome problems. We hope this is a good augury.

The Committee on Postgraduate Instruction conducted a very successful assembly for the third successive year. Again rain interfered, and it is planned to hold the Assembly earlier this year in the hope of better weather. The papers were excellent, and the question-and-answer period at luncheon was particularly worth while. The district courses have been continued, but the attendance at some might have been better.

The Committee on State and National Legislation has had a busy year. The major portion of its work is over, however, for with the exception of the annual-registration bill, the various premarital blood-test bills, the nurses' bill and some recently entered bills dealing largely with defense, the final reports are in.

Not every bill we favored was passed, nor was every bill we opposed turned down; but the important ones have gone our way, or at least have been put over for another two years. The Chiropractic Bill was beaten, and the Osteopathic Bill was withdrawn by the proposers, as was the bill to study the practice of surgery. The two bills to postpone and nullify the operation of the Approving Authority were given leave to withdraw.

Along with the Committee on Public Relations, and the Special Committee, the committee has spent a great deal of time and effort guiding our own enabling act through the Legislature. It was favorably reported by the Committee on Insurance and the Committee on Ways and Means, it passed the House, and yesterday it was passed by the Senate.

The hearings before the Committees were well attended, and I take this occasion to express the thanks of the Society to those members who gave their time to this duty.

I also thank the district chairmen for their splendid co-operation in the work of the Committee on State and National Legislation, and particularly the members of the Legislature, who gave the bill so much thought and so patiently listened to our arguments.

I am sure you all appreciate the time-consuming, exacting work that Dr. Lanman and his committee have given to this problem. Much now remains to be done to set up the organization, and it cannot be done without your hearty co-operation.

The *Journal* has continued to improve in quality and in circulation. It is interesting to note that there has been an increase of over 1000 outside subscribers – that is, nonmembers of the Massachusetts Medical Society – in the last two years. Sixty-eight per cent of the papers presented to the editorial board were accepted, an increase of 6 per cent over the preceding year. The operating loss of $20,438 is covered by your contributions to the *Journal*, which this year totaled $20,500. I am sure we have every reason to be proud of the *Journal* and its very efficient editor.

I take this occasion to thank the editor for his willingness to undertake the additional duties of secretary *pro tempore*, a service that I am sure you all join with me in appreciating.

The Council, after thorough deliberation and debate, has recommended very comprehensive changes in the by-laws, which you have today approved. The setting up of the office of president-elect will, I believe, be of great benefit, in that it offers a year of apprenticeship, so to speak. The president will come into office with a far better knowledge of the Society and its component parts than is at present possible. The Executive Committee of the Council will prove to be a more and more useful piece of machinery as time goes on. Composed of one member from every district society it will be a truly representative body. The president has to decide many questions which arise between meetings of the Council that might better be referred to a representative body such as the Executive Committee will be. The by-laws now allow the appointment by the Executive Committee of an executive secretary, if and when the affairs of the Society seem to require such a full-time officer.

It seems to me that the time has come when the headquarters of the Society should assume a certain amount of dignity. It should offer hospitality to visitors and a wholesome welcome to our members. Here should be found daily lists of clinics and meetings. It should be modeled, perhaps, something like the Academy of Medicine in New York City, manned by a competent staff and open daily throughout the week. This might mean some structural rearrangement, but I believe it could be accomplished and in the doing add to the prestige of the Society. A full-time executive secretary would amplify this scheme. I commend it to your earnest consideration.

The changes in the by-laws relating to the admission of members deserve your careful reading. The requirements for admission to fellowship by an applicant who is a graduate of a foreign or substandard domestic medical school have been greatly strengthened. In the first place he must submit the name of a fellow who agrees to be his sponsor. In the second place a local board of membership is set up in each district consisting of the president, secretary and supervising censor, who must personally interview the applicant, weigh the evidence, form an opinion and report it to the Committee on Medical Education. This committee shall then decide whether the candidate shall be examined by the censors.

The machinery for the readmission of members has been somewhat simplified and at the same time strengthened. The time-consuming method of appointing special committees to consider applications for reinstatement has been abolished and the responsibility placed in the hands of a membership committee of each district society, whereas

the ultimate authority to recommend still resides in the Committee on Membership, and the Council, as heretofore, alone has power to readmit. The thanks of the Society are due to the members of the Committee on Medical Education for their very thorough work.

Your society has taken an active part in the preparedness program. Your members have responded loyally to serve on draft, advisory and appeal boards, giving generously of their time and strength without thought of compensation. The State Committee, headed by Dr. Fitz, through the district committees has helped to round up delinquent questionnaires. It has given such aid as it could to the Army-Corps surgeons and stands ready to render more when required. It co-operated with the three medical schools of Boston in sponsoring a meeting of the draft-board and appeal-board chairmen in an effort to persuade them that the education of medical students must not be interrupted until at least one year of internship shall have been completed and to urge them to defer medical students for this period. It is gratifying to note that the Selective Service Board has finally decreed that this is a proper procedure, provided each case is acted on individually and that the student gives fair promise of becoming a reasonably good doctor.

Last June the Surgeon General of the Army proposed to the House of Delegates of the American Medical Association that they set up a committee to aid in the procurement of medical officers, recognizing that in the event of a national emergency it would be very necessary to conserve the medical profession and prevent the stripping of rural and isolated communities. This committee has been actively at work since then. Among other things we were asked to help in providing specialists for the various 1000-bed hospitals in the newly constructed camps. Lists of such available specialists were assembled and sent to the various Army-Corps surgeons within a short time. The General Staff, however, ordered that these specialists should be taken only from the Medical Reserve Officers Corps. The General Staff further decreed that no doctor should be newly commissioned in the Reserve Officers Corps above the rank of first lieutenant. It is therefore impossible for the Corps-Area surgeons to obtain all the specialists they desire. It is hoped that some way may be found to utilize available and willing specialists and to give them a rank of captain or major, commensurate with the position and authority.

The Army estimates that its reserve corps will be exhausted by the middle of summer. It also estimates that it will need a yearly turnover of something like 9000 doctors. It seems wise at present that any unmarried doctor within the draft age should apply for a reserve officer's commission, rather than be drafted to an uncertain assignment. What lies ahead of us no one knows. We do know that the United States Army, Navy and Public Health Service need many doctors, and at the same time civilian needs must not be overlooked.

In closing I thank all the officers and committees for their untiring efforts and ready responses to my calls. Their loyalty and support have meant much to me in these somewhat trying two years.

It is difficult to find words to express to you, as members of this oldest medical society in America, my thanks for the high honor you have done me.

Dr. Phippen then introduced the orator, Dr. A. Warren Stearns, who delivered the Annual Discourse, "The Role of the Physician in a Competitive Society" (published in the May 22, 1941, issue of the *New England Journal of Medicine*).

Dr. Frank R. Ober, president for the ensuing year, was introduced and made a few remarks. The meeting was adjourned at 12:35 p.m.

Following the annual luncheon in the Swiss Room, a military symposium was held in the Sheraton Room, Dr. Dwight O'Hara presiding; the attendance was at least 500.

* * *

There were twenty-two scientific and sixty-three technical exhibits, all of which were extremely well attended. The motion-picture program, which covered both days, was so popular that sufficient seating capacity was not available.

The Ladies' Committee arranged an attractive program. On May 21, after a visit in the afternoon to the Isabella Stewart Gardner Museum, there was a dinner at the Roof Garden, Hotel Ritz-Carlton. A trip to the South Shore was made on May 22, including visits to the Dorothy Quincy House and Adams Mansion in Quincy and to the Cushing Homestead and Old Ship Church in Hingham, with luncheon at Hugo's in Scituate.

The annual golf tournament was held at the Commonwealth Country Club on May 21. Forty-two members participated, and the winners were as follows:

Low net (Burrage Bowl) — A. J. A. Campbell
Second low net — H. E. Groden
Third low net — L. F. Playse
Low gross — E. E. O'Neil
Second low gross — Morgan Sargent
Third low gross — T. E. Dinan

The total registration was as follows:

Members and guest physicians	1525
Ladies	175
Total	1700

The lists of admissions and deaths are appended, together with the official lists of officers, standing and special committees, delegates, councilors, censors, section officers and officers of the district medical societies.

ROBERT N. NYE, *Secretary* pro tempore

APPENDIX

ADMISSIONS RECORDED FROM MAY 22, 1940, TO MAY 22, 1941

YEAR OF ADMISSION	RESIDENCE	MEDICAL COLLEGE
1941	Abrams, Herbert, Lowell	12
1940	Adams, Edward Everett, Roslindale	10
1941	*Adler, Alexandra, Boston	52
1941	Allan, Malcolm Stuart, Palmer	12
1940	Allen, Edward Carson, Brookline	12

1940	Anderson, Bertha Olive, Pittsfield	23
1941	Appleyard, Arthur Edward, Barre	12
1940	Bachrach, Samuel, Worcester	12
1941	Bayles, Theodore Bevier, Jamaica Plain	11
1941	Bell, George Olaf, Waban	11
1941	*Bergmann, Louis, Brighton	52
1941	*Berl, Adolf, Brighton	27
1941	*Beuthner, Bernard Hardy, Middleboro	48
1940	Bischoff, Arthur Jacob, Stoneham	28
1940	Bissell, George DeForest, Jr., Melrose	10
1941	Bliss, Sheldon Pratt, East Braintree	12
1940	Bowen, Donald Eyre, West Newton	12
1940	Boyer, Norman Howard, Lexington	12
1941	*Brugsch, Heinrich Georg, Belmont	49
1940	*Burger, Francis Joseph, Jr., Watertown	6
1941	Burke, Arthur Edward, East Gardner	12
1940	Burke, Edward William, Brighton	12
1941	Butler, Henry Rutherford, Quincy	11
1941	Butterfield, Paul Morgan, Harwich Center	1
1941	Cahill, Francis Paul, Cambridge	11
1940	*Camoesas, Joao Jose Da Conceicao, Taunton	46
1940	Campbell, Harry Lincoln, Medford	10
1941	Carey, Edward Gerald, Boston	12
1941	Cary, Francis Frothingham, Springfield	11
1940	Charron, Joseph Rosario, Turners Falls	37
1940	Chase, Winnifred Pingree, West Medford	12
1940	Chesbro, Wallace Leo, Springfield	12
1940	Chisholm, Julian Ford, Jr., Boston	18
1941	Coco, Joseph Paul, Cheshire	43
1940	Cole, Ralph Earle, Westford	12
1941	Collinson, Arthur William, Boston	12
1941	Compson, James Enoch, Worcester	11
1941	Conlin, John Francis, Chelsea	12
1940	Connolly, Richard Francis, Danvers	19
1941	Cordray, David Price, Boston	22
1940	Crehan, Paul Joseph, Woburn	7
1941	*Cunningham, Joseph Anthony, Marblehead	30
1941	Dalrymple, Leolia Agnes, Boston	32
1940	*de Aguiar, Alcinda Pereira, Wrentham	55
1940	Dee, John Edwin, Worcester	11
1940	Deming, Julia, Boston	44
1940	Denhoff, Eric, Boston	7
1941	Denton, Joseph Howard, Jr., Falmouth	24
1941	*Deutsch, Helene, Cambridge	52
1941	Dick, Vernon Shelton, Waban	38
1941	Dieuaide, Francis Raymond, Boston	18
1941	Dingle, John Holmes, Boston	11
1940	Dixey, Grant Milton, Marblehead	10
1941	Doyle, Bernard Joseph, Easthampton	12
1940	Dutton, David Parkhurst, Stoneham	11
1940	Edsall, Geoffrey, Cambridge	11
1940	*Elsberg, Albert, Brookline	49
1941	Evans, James Ambrose, Waban	11
1941	*Faillace, Fedele Michele, Brookline	56
1940	Farrington, Robert F., Boston	11
1940	Fell, Ernest Millward, Boston	12
1941	*Fischmann, Joseph, Brighton	49
1941	Fisk, Evelyn Bernice, West Newton	7
1940	*Fleischner, Felix George, Greenfield	52
1940	*Fleury, Edgar Joseph, Holyoke	6
1940	Foster, Frank Pray, Newton	9
1940	Friend, Dale Gilbert, Boston	11
1940	Garber, Israel Enoch, Pittsfield	10
1941	Gardner, George Edward, Belmont	11
1940	Girouard, Fernand L., Attleboro	12
1940	Gladstone, Robert Warren, Pittsfield	7
1941	Goehring, Harrison Dilwyn, Newton Upper Falls	47
1941	*Goldstein, Kurt, Boston	48
1941	*Goldstein, Walter, Brockton	49
1940	*Gordon, Minerva, Attleboro	6
1941	Goulston, Harold Francis, New Bedford	12
1940	*Grace, James Joseph, Springfield	6
1940	Grassi, Michael Anthony, Haverhill	12
1940	Gratwick, Mitchell, Andover	18
1940	Greene, Daniel Crosby, Jr., Pittsfield	1
1940	*Grossman, Samuel, Roxbury	40
1940	Hall, Volta Ross, Jr., Arlington Heights	42
1940	Hamilton, Thomas Stewart, Truro	13
1940	Harrington, Philip Vincent, Worcester	11
1941	Haskell, Helen Stevens, Northampton	33
1941	*Hauptmann, Alfred, Brighton	45
1941	Hayes, Donald Robert, Waltham	11
1940	*Heavey, Thomas J., Medway	6
1941	Hertz, Saul, Brookline	11
1941	*Hess, Leo, Brookline	52
1940	Higgins, Raymond Francis, Worcester	9
1940	*Hirsch, Oskar, Boston	52
1940	Hoerr, Stanley Obermann, Brookline	11
1940	*Hollander, Alfred, Springfield	26
1940	Hollis, Charles Hilliard, Lawrence	12
1940	Horne, Elwood Otho, Shrewsbury	12
1940	Howard, Paul Malleville, Waverley	22
1940	Huntington, Benjamin Lincoln, Boston	11
1940	*Igersheimer, Josef, Boston	49
1941	Ingelfinger, Franz Joseph, Brighton	11
1940	Isherwood, Elizabeth Pynchon, Westfield	12
1941	*Izen, David Sidney, Medford	6
1941	*Jessner, Lucie Johanna Ney, Georgetown	3
1941	*Jossmann, Paul Bernhard, Brookline	54
1940	Jovell, John William, New Bedford	12
1941	*Kahn, Trude, Springfield	26
1940	*Kant, Otto, Shrewsbury	35
1940	Kasparian, Karl Der, Boston	12
1940	Koczera, Stanley Joseph, New Bedford	12
1941	Kranichuck, John Michael, South Hadley Falls	12
1941	Kubik, Charles Soucek, South Lincoln	15
1940	*Kwalski, Joseph Vincent, Ware	6
1940	Lake, Fredric David, Springfield	11
1941	Lamphier, James Andre, Newton	12
1941	Lane, Virginia Elizabeth, East Northfield	44
1940	Larson, Carroll Bernard, Brookline	61
1941	Lawrence, Knowles Birkinshaw, Brookline	1
1940	Lendgren, Carl Victor, Foxboro	10
1941	*Lepehne, Georg, Brookline	3
1940	Lepore, John James, Marlboro	19
1941	*Levinger, Max D., Fitchburg	31
1940	Lewis, Elizabeth Katherine, Groton	12
1940	Lewis, F. Woodward, Groton	12
1940	Lichter, Gerald Israel, Boston	14
1941	Lowell, Francis Cabot, Cambridge	11
1941	*Lowenthal, Frederick, Brookline	26
1940	*Lowenthal, Karl, Brighton	30
1941	*Lowenthal, Theresa Ulla, Brighton	49
1941	Lowis, Samuel, Brookline	11
1941	*Luisada, Aldo Augusto, Brookline	17
1940	MacDonald, Wilfred Daniel, Worcester	12
1941	Marcus, Phillip Stephen, Boston	2
1941	*Marnell, Thomas Frank, Great Barrington	8
1941	*Martin, Donald Alton, Waltham	5
1941	*Marx, Martin Simon, Brockton	31
1940	*Mastrangelo, Louis, Watertown	6
1941	*Mathews, Molyneaux Paul, Brockton	6
1941	*Mautner, Hans, Waltham	52
1941	McDaniel, Lewis Tillman, Boston	11
1941	McDermott, Leo James, Boston	11
1941	McDonald, Eugene Joseph, Boston	12
1941	McFadden, James Marshall, Jr., Jamaica Plain	36
1940	McGee, Charles Joseph, Brockton	11
1940	*McSweeney, Timothy Hubert, Wilbraham	6

1941	Meany, John Hartley, Worcester	9
1941	Memery, Harry Noel, Longmeadow	12
1940	*Meyersohn, Franz, Worcester	49
1940	*Michelsen, Jost Joseph, Winchester	49
1940	*Michelson, Harry Hirsch, Northampton	4
1940	Miller, Carroll Cameron, Boston	11
1940	Miller, Wallace Charles, Brookline	16
1940	*Molliver, Henry, Chelsea	6
1941	Morris, Soli, North Grafton	51
1940	Morse, William Jordan, Jr., Attleboro	12
1941	*Mortara, Franco, Somerville	20
1940	*Moulyn, Adrian Cornelis, Topsfield	21
1940	*Mueller, Hans Peter, Cambridge	3
1941	Mullin, Charles Samuel, Jr., Dorchester Center	12
1941	*Nathan, Paul, Holyoke	30
1941	*Nauen, Alice, Brighton	63
1941	*Nothmann, Martin Manheiur, Brighton	48
1940	*Oppler, Willy, Hathorne	49
1940	Paddock, Franklin Kittredge, Pittsfield	11
1941	Patton, William Ernest, North Grafton	25
1940	*Pavenstedt, Eleanor, Boston	54
1941	†Pearlstein, Max, Braintree	12
1940	Pellerin, Alcide Damase, New Bedford	10
1940	*Pendola, Anthony Salvatore, Holyoke	6
1940	*Perona, Attilio Charles, Norwood	6
1940	Pike, George Manuel, Boston	11
1941	Pinto, Sherman Spalding, Arlington	34
1941	Pippitt, Richard B., Cambridge	11
1940	*Pollen, David Anopolsky, Allston	6
1941	*Por, Frieda, Brookline	52
1941	*Poras, Harry Hermann, Clinton	52
1940	Potts, William Law, Boylston	42
1940	Pratt, Gordon Carver, Longmeadow	15
1941	*Presser, Karl, Boston	52
1941	Putnam, Marian Cabot, Cambridge	18
1941	Reeves, Dorothy Mae, Worcester	38
1941	*Reinauer, Anna Jeanette, Brookline	39
1941	Rhees, Morgan John, Boston	11
1941	Riley, Joseph Gerard, Waltham	12
1941	*Ritvo, Meyer, Watertown	6
1940	Roberts, Charles Purcell, Boston	41
1940	Rooney, Charles Edwin, Woburn	12
1940	*Rosenblum, Harry Aaron, Fiskdale	40
1940	Ross, Ralph Adolph, Brighton	11
1940	Rothblatt, Bernard William, Jamaica Plain	12
1940	Rubin, Harold William, Chelsea	12
1941	*Ruesch, Jurgen, Cambridge	29
1940	Ryan, Vincent Gerard, Georgetown	1
1940	*Salomon, Robert, Worcester	50
1941	*Scherman, Richard Paul, South Hanson	31
1940	*Schott, Edward Francis, Lynn	53
1940	Schulz, Milford David, Boston	57
1940	Seale, Earl Spinks, Boston	58
1940	Segel, Arnold Lester, Brighton	11
1941	Seigal, Harold Leonard, Boston	59
1941	Sheridan, Philip Henry, Northampton	9
1940	Shwachman, Harry, Roxbury	18
1941	*Singer, Karl, Allston	52
1941	Smith, Eugene Francis, Milford	12
1940	Snow, Robert Groesbeck, Winchester	11
1940	*Spelios, Louis George, Springfield	6
1941	*Spiegl, Erwin David, Boston	62
1940	Sprague, John Stabler, Brighton	18
1940	Spring, George Viner, East Pepperell	12
1941	Stead, Eugene Anson, Jr., Boston (Roxbury)	41
1940	Stenzel, Franz Robert, Waban	11
1940	Stevens, Herman David, Boylston	59
1941	Suby, Howard Ingram, Brookline	11
1941	*Sulzberger, Fred Max, Southbridge	31
1940	Svibergson, Nils Emil, Tewksbury	12
1940	Sweetser, Peter Wilbur, Boston	19
1941	*Thimann, Joseph, Cambridge	52
1940	Thomas, Edwin Bentley, Tewksbury	12
1941	Tuohy, Edward Lawrence, Cambridge	60
1941	Ulfelder, Howard, Boston	11
1940	Verstandig, Charles Coleman, Fort Devens	2
1941	*von Bruecke, Dora, Roxbury	52
1940	*Wagner, Richard, Boston	52
1940	Wassersug, Joseph David, Middleboro	12
1940	Weiss, William George, Methuen	9
1940	*Welch, Richard Francis, Milton	6
1940	Wheeler, Peter William, Haverhill	12
1941	White, George, Wrentham	12
1941	White, Roland Hubert, Stoneham	28
1940	Whitelaw, George Percy, Boston	11
1941	Wilkins, Robert Wallace, Brookline	11
1940	Williams, Robert Hardin, Quincy	18
1941	Woods, William Leon, Boston	32
1941	Yavarow, Millin Mully, Lowell	12
1940	Yeomans, Andrew, Brookline	11
1940	*Young, Alexander, Hyde Park	6
1940	Young, David Alexander, Waverley	11
1941	*Zaritt, Hyman I., Gilbertville	6
1941	*Zaudy, Eleanore Caecilie, Roxbury	64
1940	Zentgraf, Leo Paul, Manchester	10

Total number of new fellows admitted November, 1940	126
Total number of new fellows admitted May, 1941	112
Total number of fellows readmitted May, 1941	1
Grand total	239

*The candidate's diploma was approved by the Committee on Medical Education and Medical Diplomas, and he or she was given a personal interview by this committee and permitted to take an examination before a board of censors.

†Readmitted by censors.

KEY TO MEDICAL COLLEGES

1. Yale University School of Medicine
2. University of Tennessee College of Medicine
3. University of Königsberg
4. University of Kiel
5. University of Edinburgh
6. Middlesex University School of Medicine
7. University of Vermont College of Medicine
8. Missouri College of Medicine and Science
9. McGill University Faculty of Medicine
10. Boston University School of Medicine
11. Harvard Medical School
12. Tufts College Medical School
13. Wayne University College of Medicine
14. Vanderbilt University School of Medicine
15. Rush Medical College of the University of Chicago
16. Loyola University School of Medicine
17. Royal University Medical School, Florence, Italy
18. Johns Hopkins University School of Medicine
19. Georgetown University School of Medicine
20. University of Bologna
21. University of Utrecht
22. University of Pennsylvania School of Medicine
23. University of Kansas School of Medicine
24. Long Island College of Medicine
25. George Washington University School of Medicine
26. University of Bonn
27. University of Leipzig
28. College of Medical Evangelists
29. University of Zurich

30. University of Freiburg
31. University of Munich
32. University of Toronto Faculty of Medicine
33. Cornell University Medical College
34. University of Nebraska College of Medicine
35. University of Göttingen
36. Indiana University School of Medicine
37. Laval University Faculty of Medicine
38. University of Michigan Medical School
39. Friedrich-Alexanders University
40. Kansas City University of Physicians and Surgeons
41. Emory University School of Medicine
42. Western Reserve University School of Medicine
43. University of Virginia Department of Medicine
44. Woman's Medical College of Pennsylvania
45. University of Heidelberg
46. Lisbon University Faculty of Medicine
47. University of Pittsburgh School of Medicine
48. University of Breslau
49. University of Berlin
50. University of Frankfort
51. New York University College of Medicine
52. University of Vienna
53. University of Strassburg
54. University of Geneva
55. Oporto University, Portugal
56. University of Rome
57. Northwestern University Medical School
58. Tulane University of Louisiana School of Medicine
59. Jefferson Medical College
60. University of Minnesota Medical School
61. State University of Iowa College of Medicine
62. University of Prague
63. University of Hamburg
64. University of Bern

Deaths Reported from May 22, 1940 to May 22, 1941

ADMITTED	NAME	PLACE OF DEATH	DATE OF DEATH	AGE
1907	Andrews, Frederick Francis	Revere	November 7, 1940	68
1920	Begg, Alexander Swanson	West Roxbury	September 26, 1940	59
1875	†Benner, Burnham Roswell	Lowell	May 29, 1940	93
1904	Binford, Ferdinand Augustus	Hyannis	September 1, 1940	67
1912	†Blodgett, Stephen Haskell	South Lincoln	September 3, 1940	76
1933	Booth, Ernest Lazarus	East Boston	March 24, 1941	57
1887	†Bowen, John Templeton	Boston	December 3, 1940	83
1899	Briggs, Lloyd Vernon	Tucson, Arizona	February 28, 1941	78
1897	Carleton, Ralph	Springfield	October 26, 1940	70
1940	Chippendale, Francis David John	Fall River	November 19, 1940	28
1886	Clark, Joseph Payson	Boston	July 21, 1940	80
1926	Cleaves, Edwin Nelson	Boston	February 24, 1941	51
1895	Codman, Ernest Amory	Ponkapoag	November 23, 1940	71
1929	Copeland, Stanley Earle	Worcester	May 13, 1941	39
1912	Cotter, Timothy Francis	Haverhill	July 3, 1940	58
1933	Cox, Clyde Edward	Worcester	November 10, 1940	35
1920	Crawford, Joseph Warrington	North Adams	November 7, 1940	68
1903 } 1920 }	Crofts, Nicholas Matthew	North Adams	September 17, 1940	75
1904	Dailey, Edward Joseph	Somerville	December 10, 1940	61
1908	Darling, Arthur Edwin	Boston	June 19, 1940	62
1899	Davis, Charles Henry	South Hamilton	January 14, 1941	67
1907	†Davis, Frank Albert	Washington, D. C.	October 7, 1940	74
1934	Decker, John Joseph	Sioux City, Iowa	December 18, 1940	36
1886	†Dewey, Charles Gipson	Dorchester	April 18, 1941	81
1900	Elliot, Henry Libbey	Thomaston, Maine	June 27, 1940	66
1917	Emerson, Frederick Lincoln	Boston	November 10, 1940	78
1896 } 1919 }	†Enebuske, Claës Julius	Lund, Sweden	July 4, 1940	85
1929	Ferrini, Peter	Laconia, New Hampshire	August 5, 1940	39
1933	Fitzpatrick, Francis Joseph	Somerville	January 2, 1941	59
1891	†Francis, Carleton Shurtleff	Kerrville, Texas	March 8, 1941	75
1890	†Goodman, Samuel	Boston	May 28, 1940	75
1901	Greene, Daniel Crosby	Newton	April 4, 1941	68
1906	†Halsall, Mary Elizabeth	Winthrop	August 1, 1940	75
1908	Harmer, Torr Wagner	Winchester	October 2, 1940	59
1898	Harvey, William Wirt	Boston	November 10, 1940	74
1914	Hayes, William Francis	Beverly	April 4, 1940	58
1885	Henry, John Goodrich	Winchendon	January 18, 1941	83
1897	Hewitt, Clarence Elbert	Springfield	December 18, 1940	78
1904	Hill, George Jackson	Boston	October 29, 1940	61
1890	†Hitchcock, Henry Russell	Plymouth	November 23, 1940	79
1919	Hooper, George Henry	Belmont	July 24, 1940	48
1915	†Hoyt, Edward Malcolm	Georgetown	March 28, 1941	83
1894	†Hubbard, Osmon Huntley	Gilsum, New Hampshire	January 5, 1941	79
1891	Hutchings, Joseph Henry	Woburn	January 12, 1941	77
1884	Jackson, Henry	Chestnut Hill	October 4, 1940	82

1891 } 1920 }	Jenkins, Thomas Lincoln	Topsfield	July 29, 1940	74
1932	Kramer, Florence	Miami, Florida	January 15, 1941	56
1899	†Latham, Benoni Mowry	Taunton	July 24, 1940	67
1894	Lovell, David Bigelow	Worcester	September 18, 1940	75
1933	Magee, Edward Joseph	Danvers	January 20, 1941	77
1913	Mason, Gilbert McClellan	Dorchester	March 17, 1941	69
1928	Matzek, Neil Clayton	Belmont	May 13, 1941	45
1890	†McCarthy, Thomas Horatio	Brockton	April 11, 1941	77
1897	†Mead, Frederick Ammi	Chicopee	November 14, 1940	71
1906	†Mellen, Eleanor Way-Allen	Newton Highlands	April 19, 1941	72
1905	Mellus, Edward	Newton	December 7, 1940	68
1907	Moran, Charles Leo	Brighton	November 5, 1940	61
1933	Muir, Laura Helen	Roslindale	August 19, 1940	58
1916	Muldoon, Mary Theresa	Lexington	July 18, 1940	46
1914	Murphy, Edward Frederick	Jamaica Plain	January 15, 1941	64
1905	Murphy, Frederick Paul	Lowell	April 26, 1941	60
1905	†Murphy, Thomas William	Lawrence	November 2, 1940	70
1934	Murray, Michael Earley, Jr.	Cambridge	April 21, 1941	36
1920	Nowell, Howard Wilbert	Brookline	October 28, 1940	68
1914	O'Rourke, Edward James	Cambridge	November 20, 1940	54
1894	Ott, George John	Boston	March 29, 1941	73
1913	Packard, Loring Bradford	Brockton	April 12, 1941	64
1898	Pagé, Joseph Gregory Elias	Southbridge	December 28, 1940	69
1914	Parker, Raymond Brewer	Winthrop	October 19, 1940	55
1891	Pease, Edward Allen	Pasadena, California	June 19, 1940	75
1909	Pillsbury, Fritzroy Farnsworth	Saco, Maine	June 18, 1940	55
1873 } 1877 }	†Porter, Francis Edward	Auburndale	October 11, 1940	96
1922	Renaud, Ulric Joseph	Brockton	August 12, 1940	43
1891	†Richardson, Anna Gove	Roslindale	February 1, 1941	80
1913	†Richardson, Oscar	Roslindale	August 28, 1940	80
1890	Rogers, Albert Edward	Boston	August 8, 1940	72
1883	†Rogers, Frank Alvin	Roxbury	November 15, 1940	85
1919	Ruggles, Edwin Pakenham	Dorchester	June 19, 1940	77
1895	St. Clair, Austin Emery	Framingham	August 7, 1940	75
1881	Sanborn, Perley Lewis	Marblehead	February 3, 1941	90
1884	Sears, George Gray	Boston	May 28, 1940	81
1895	†Shattuck, Albert Milo	Worcester	May 26, 1940	69
1902 } 1912 }	Shea, Peter Owen	Worcester	March 9, 1941	70
1914	Simmons, Lt.-Col. Ralph Hayward	Washington, D. C.	March 13, 1941	52
1895	Stanley, Josiah Murch	Northboro	June 4, 1940	80
1928	Sutherland, John Preston	Boston	February 21, 1941	87
1908	Swan, Lawrence Clarke	Beverly	October 2, 1940	59
1882	†Swan, Roscoe Wesley	Worcester	October 13, 1940	80
1895	Sweet, Frederick Benoni	Springfield	May 10, 1941	70
1887	Thorndike, Augustus	Bar Harbor, Maine	August 23, 1940	77
1938	Villone, Anthony Joseph	Quincy	October 6, 1940	49
1917	Vivian, William James	Northport, New York	June 10, 1940	56
1915	Walker, Melvin Harvey, Jr.	Pittsfield	November 21, 1940	54
1913	Walsh, Edmund Francis	Nahant	August 20, 1940	56
1880	†Walton, George Lincoln	Boston	January 17, 1941	87
1905	Warren, Franklin Lafayette	Bridgewater	February 11, 1941	69
1890	Weeks, Joshua Franklin	New Bedford	January 12, 1941	74
1923	Welles, Delbert Arthur	Fitchburg	May 1, 1941	76
1919	Wetherell, Bryant David	Boston	March 5, 1941	52
1900	Whoriskey, John Joseph	Cambridge	July 26, 1940	63
1916	Williams, Frederick Horace	Boston	December 6, 1940	67
1879 } 1897 }	†Woods, George Lyman	Knoxville, Tennessee	July 28, 1940	90
1901	Wormelle, Charles Burton	Allston	September 16, 1940	68
1909 } 1933 }	†Worthing, Frank Bertelle	Chatham	March 13, 1941	71

†Retired fellow.

Total number of deaths of active fellows	80
Total number of deaths of retired fellows	24
Grand total	104

Officers for 1941-1942

Elected by the Council May 21–22, 1941

President: Frank R. Ober, Boston, 234 Marlboro Street.
President-Elect: George L. Schadt, Springfield, 44 Chestnut Street.
Vice-President: Edward P. Bagg, Holyoke, 207 Elm Street.
Secretary: Michael A. Tighe, Lowell. Office, Boston, 8 Fenway.
Treasurer: Charles S. Butler, Boston, 257 Newbury Street.
Orator: William B. Castle, Boston, Boston City Hospital.

Standing Committees for 1941-1942

Elected by the Council, May 21-22, 1941

Date of Appointment

Publications — Established 1825.

R. M. Smith	June 6, 1933 (appointed chairman May 21, 1941)
J. P. O'Hare	June 9, 1936
Conrad Wesselhoeft	June 2, 1937
W. B. Breed	February 7, 1940
Oliver Cope	May 21, 1941

Arrangements — Established 1849.

W. T. O'Halloran	June 2, 1937 (appointed chairman May 1, 1941)
J. A. Halsted	June 1, 1938
G. P. Sturgis	June 7, 1939
H. H. Faxon	May 21, 1940
G. M. Morrison	May 21, 1941

Ethics and Discipline — Established 1871.

R. R. Stratton	June 9, 1936 (appointed chairman May 21, 1941)
W. J. Brickley	February 3, 1937
A. G. Rice	June 1, 1938
F. R. Jouett	May 21, 1940
A. R. Gardner	May 21, 1941

Medical Education — Established 1881.

J. P. Monks	June 7, 1939 (appointed chairman May 21, 1940)
A. W. Stearns	June 9, 1936
G. D. Henderson	June 1, 1938
L. S. McKittrick	May 21, 1940
R. T. Monroe	May 21, 1941

State and National Legislation — Established 1894.

H. C. Marble	October 2, 1940 (chairman)
D. L. Lionberger	June 4, 1935
B. F. Conley	June 2, 1937
C. A. Robinson	June 1, 1938
E. M. Chapman	June 7, 1939

Membership — Established 1897.

G. C. Caner	June 17, 1930 (appointed chairman May 21, 1940)
J. E. Fish	June 17, 1930
H. F. Newton	June 9, 1931
P. H. Leavitt	June 1, 1938
A. W. Reggio	May 21, 1940

Public Health — Established 1912.

F. P. Denny	June 1, 1938 (appointed chairman June 7, 1939)
Gerald Hoeffel	June 17, 1930
S. C. Dalrymple	June 4, 1935
H. L. Lombard	June 4, 1935
H. F. Day	June 7, 1939

Medical Defense — Established 1927.

A. W. Allen	June 7, 1927 (appointed chairman June 7, 1939)
E. D. Gardner	June 7, 1927
W. R. Morrison	June 9, 1936
Horatio Rogers	June 7, 1939
G. S. Reynolds	May 21, 1941

Permanent Home — Established 1932.

W. H. Robey	February 24, 1937 (chairman)
C. G. Mixter	June 8, 1932
J. M. Birnie	June 8, 1932
C. S. Butler	June 4, 1935
E. C. Miller	June 4, 1935

Financial Planning and Budget — Established 1938.

John Homans	June 2, 1938 (chairman)
E. L. Hunt	June 2, 1938
C. F. Wilinsky	June 2, 1938
E. J. O'Brien, Jr.	June 2, 1938
P. P. Johnson	October 4, 1939

Special Committees

Cancer — Established 1917.

Shields Warren, chairman; F. G. Balch, E. M. Daland, P. E. Truesdale, C. C. Simmons.

Representatives to the Massachusetts Central Health Council

Barnstable: W. D. Kinney*
Berkshire: R. J. Carpenter*
Hampden: G. D. Henderson*
Norfolk: F. P. Denny*
Suffolk: R. B. Osgood*
Worcester: E. C. Miller*

Public Education (a subcommittee of the Committee on Public Health) — Established 1930.

F. P. Denny, chairman; Gerald Hoeffel, secretary; G. R. Minot, W. H. Robey, R. M. Smith, E. H. Place, C. C. Simmons, J. H. Pratt, H. W. Stevens, J. B. Ayer, H. P. Mosher, F. R. Ober, E. P. Joslin, J. D. Barney, H. L. Lombard.

Public Relations — Established 1931. (One member appointed yearly by each district medical society; the president of the Society is chairman.)

Barnstable District Medical Society

M. E. Champion, North Harwich.

Berkshire District Medical Society

P. J. Sullivan, Dalton, 471 Main Street.

Bristol North District Medical Society

J. H. Brewster, Attleboro, 178 South Main Street.

Bristol South District Medical Society

A. J. Pothier, New Bedford, 720 County Street.

*Interim appointment.

ESSEX NORTH DISTRICT MEDICAL SOCIETY

E. S. Bagnall, Groveland, 281 Main Street. (Secretary of committee.)

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

E. D. Reynolds, Danvers, 48 High Street.

FRANKLIN DISTRICT MEDICAL SOCIETY

H. G. Stetson, Greenfield, 39 Federal Street.

HAMPDEN DISTRICT MEDICAL SOCIETY

P. E. Gear, Holyoke, 188 Chestnut Street.

HAMPSHIRE DISTRICT MEDICAL SOCIETY

L. N. Durgin, Amherst, 66 Amity Street.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

J. H. Blaisdell, Winchester. Office, Boston, 45 Bay State Road.

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

D. J. Ellison, Lowell, 8 Merrimack Street.

MIDDLESEX SOUTH DISTRICT MEDICAL SOCIETY

D. C. Dow, Cambridge, 1587 Massachusetts Avenue.

NORFOLK DISTRICT MEDICAL SOCIETY

N. A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Avenue.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

F. A. Bartlett, Wollaston, 308 Beale Street.

PLYMOUTH DISTRICT MEDICAL SOCIETY

C. D. McCann, Brockton, 12 Cottage Street.

SUFFOLK DISTRICT MEDICAL SOCIETY

A. A. Hornor, Boston, 319 Longwood Avenue.

WORCESTER DISTRICT MEDICAL SOCIETY

C. A. Sparrow, Worcester, 73 Sagamore Road.

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

J. J. Curley, Leominster, 82 Main Street.

POSTGRADUATE INSTRUCTION — Established 1932.

Reginald Fitz, chairman*; L. E. Parkins, secretary; F. D. Adams, Roy Morgan, J. M. Birnie, H. L. Higgins, J. W. O'Connor, C. W. Blackett, Jr., R. B. Osgood, C. S. Burwell, A. W. Stearns, W. H. Robey, R. N. Nye, H. D. Chadwick, C. M. Campbell, Lincoln Davis.

PHYSICAL THERAPY — Established 1935.

F. P. Lowry, chairman; R. B. Osgood, G. R. Minot.

COMMITTEE TO CONSIDER EXPERT TESTIMONY — Established 1936.

G. L. Schadt, chairman; David Cheever, J. J. Goodwin, F. P. McCarthy, H. C. Marble.

AUTOMOBILE INSURANCE CLAIMS — Established 1937.

H. C. Marble, chairman; H. M. Landesman, secretary; P. P. Henson.

CONVALESCENT CARE — Established 1938.

T. D. Jones, chairman; H. E. Gallup.

INDUSTRIAL HEALTH — Established 1939.

W. I. Clark, chairman; L. R. Daniels, D. L. Lynch,* H. G. Murray, T. L. Shipman.

*Interim appointment.

ARMY MEDICAL LIBRARY AND MUSEUM — Established 1939.

H. R. Viets, chairman; R. B. Osgood, Benjamin Spector.

COMMITTEE TO STUDY PRACTICE OF MEDICINE BY UNREGISTERED PERSONS — Established 1939.

Richard Dutton, chairman; B. F. Conley, E. F. Timmins.

TWENTY-FIVE VOTING MEMBERS IN THE ASSOCIATED HOSPITAL SERVICE CORPORATION — Established 1939.

B. H. Alton, E. S. Bagnall, G. M. Balboni, W. B. Breed, L. D. Chapin, H. F. Day, J. F. Donaldson, A. W. Dudley, J. M. Fallon, J. E. Flynn, A. R. Gardner, H. W. Godfrey, D. C. Halbersleben, J. A. Halsted, J. H. Lambert, A. A. Levi, A. E. Parkhurst, Helen S. Pittman, A. G. Rice, A. T. Ronan, F. W. Snow, G. L. Steele, R. P. Stetson, J. E. Talbot, E. L. Young.

COMMITTEE TO STUDY PROPOSALS FOR BUDGETING MEDICAL CARE — Established 1940.

J. C. McCann, chairman*; E. S. Bagnall,* J. M. Birnie, T. H. Lanman, Shields Warren.

TAX-SUPPORTED MEDICAL CARE COMMITTEE — Established 1940.

E. S. Bagnall, chairman; A. L. Duncombe, A. A. Hornor, E. L. Hunt, W. J. Pelletier.

COMMITTEE TO MEET WITH MASSACHUSETTS HOSPITAL ASSOCIATION — Established 1940.

H. M. Clute, chairman; B. H. Alton, R. B. Cattell, E. D. Gardner, A. E. Parkhurst, F. W. Snow.

COMMITTEE ON MEDICAL PREPAREDNESS — Established 1940.

Reginald Fitz, chairman.

COMMITTEE TO EXAMINE WPA RECORDS — Established 1940.

G. L. Richardson, chairman; W. E. Browne, D. J. Ellison, L. R. Chaput, F. P. McCarthy.

MATERNAL WELFARE — Established 1941.

J. A. Smith, chairman; Thomas Almy, R. L. DeNormandie, M. Luise Diez, C. J. Duncan, M. F. Eades, A. F. G. Edgelow, Rachel L. Hardwick, J. W. O'Connor, L. E. Phaneuf, G. M. Shipton, W. R. Sisson, R. M. Smith, R. S. Titus, R. J. Williams.

COMMITTEE TO STUDY THE PRACTICE OF MEDICINE — Established 1941.

Dwight O'Hara, chairman; B. H. Alton, A. E. Parkhurst, D. D. Scannell, Conrad Wesselhoeft.

DELEGATES AND ALTERNATES TO THE HOUSE OF DELEGATES, AMERICAN MEDICAL ASSOCIATION FOR 1941-1942

DELEGATES	ALTERNATES
June 1, 1940 to June 1, 1942	
D. D. Scannell	E. S. Bagnall, Groveland
Jamaica Plain	
Dwight O'Hara, Waltham	E. L. Hunt, Worcester
C. E. Mongan, Somerville	M. A. Tighe, Lowell
W. G. Phippen, Salem	J. I. B. Vail, Hyannis

June 1, 1941 to June 1, 1943

J. M. Birnie, Springfield
R. H. Miller, Boston
R. J. Carpenter, Pittsfield
Cadis Phipps, Brookline

Executive Committee of the Council

Members Ex-Officiis

President: Frank R. Ober, Boston, 234 Marlboro Street.
President-Elect: George L. Schadt, Springfield, 44 Chestnut Street.
Vice-President: Edward P. Bagg, Holyoke, 207 Elm Street.
Secretary: Michael A. Tighe, Lowell. Office, Boston, 8 Fenway.
Treasurer: Charles S. Butler, Boston, 257 Newbury Street.

Term Expires 1942

Essex South: Loring Grimes, Swampscott, 84 Humphrey Street.
Hampshire: Lawrence N. Durgin, Amherst, 66 Amity Street.
Middlesex South: Dwight O'Hara, Waltham. Office, Boston, 5 Bay State Road.
Norfolk South: Daniel B. Reardon, Quincy, 1186 Hancock Street.
Suffolk: Augustus Thorndike, Jr., Boston, 319 Longwood Avenue.
Worcester: Ralph S. Perkins, Worcester, 10 Hackfeld Road.

Term Expires 1943

Barnstable: William D. Kinney, Osterville.
Bristol North: William H. Allen, Mansfield, 70 North Main Street.
Bristol South: Edwin D. Gardner, New Bedford, 150 Cottage Street.
Essex North: Frank W. Snow, Newburyport, 24 Essex Street.
Middlesex East: Kenneth L. Maclachlan, Melrose, 1 Bellevue Avenue.
Plymouth: Peirce H. Leavitt, Brockton, 129 West Elm Street.

Term Expires 1944

Berkshire: John J. Boland, Pittsfield, 334 North Street.
Franklin: Frederick J. Barnard, Greenfield, 479 Main Street.
Hampden: George L. Steele, Springfield, 20 Maple Street.
Middlesex North: William M. Collins, Lowell, 174 Central Street.
Norfolk: Carl Bearse, Boston, 483 Beacon Street.
Worcester North: John J. Curley, Leominster, 82 Main Street.

Councilors for 1941–1942

Elected by the District Medical Societies at Their Annual Meetings, April 15 to May 15, 1941

Barnstable
O. S. Simpson, Centerville, Main St., V. P.
M. E. Champion, North Harwich, A. M. N. C.
D. E. Higgins, Cotuit, Main St., Sec.
C. H. Keene, Chatham, Seaview St.
W. D. Kinney, Osterville, E. C., M. N. C.

Berkshire
W. A. Millet, Pittsfield, 225 North St., V. P.
J. J. Boland, Pittsfield, 334 North St., E. C.
R. J. Carpenter, North Adams, 85 Main St.
I. S. F. Dodd, Pittsfield, 34 Fenn St.
H. J. Downey, Pittsfield, 184 North St.
C. F. Fasce, Pittsfield, 311 North St., A. M. N. C.
C. F. Kernan, Pittsfield, 184 North St.
G. S. Reynolds, Pittsfield, 7 North St., Sec.
P. J. Sullivan, Dalton, 471 Main St., M. N. C.

Bristol North
J. A. Reese, Attleboro, 48 Bank St., V. P.
W. H. Allen, Mansfield, 70 North Main St., E. C., M. N. C.
J. H. Brewster, Attleboro, 178 South Main St.
R. M. Chambers, Taunton, Taunton State Hospital.
J. L. Murphy, Taunton, 23 Cedar St., A. M. N. C.
W. H. Swift, Taunton, 141 High St., Sec.

Bristol South
H. P. Sawyer, Fall River, 68 Bigelow St., V. P.
G. W. Blood, Fall River, 82 New Boston Rd.
R. B. Butler, Fall River, 278 North Main St., A. M. N. C.
E. F. Cody, New Bedford, 105 South Sixth St., M. N. C.
J. A. Fournier, Fall River, 11 Choate St.
E. D. Gardner, New Bedford, 150 Cottage St., E. C.
F. M. Howes, New Bedford, 135 Cottage St.
D. R. Mills, Edgartown.
H. E. Perry, New Bedford, 159 Cottage St.
A. H. Sterns, New Bedford, 31 Seventh St., Sec.
I. N. Tilden, Mattapoisett, Barstow St.
C. C. Tripp, New Bedford, 416 County St.
P. E. Truesdale, Fall River, 151 Rock St.

Essex North
C. W. Bullard, Newburyport, 194 High St., V. P.
E. S. Bagnall, Groveland, 281 Main St., A. M. N. C.
R. V. Baketel, Methuen, 7 Hampshire St.
L. R. Chaput, Haverhill, 3 Washington Sq.
E. H. Ganley, Methuen, 251 Broadway.
H. R. Kurth, Lawrence, 477 Essex St., Sec.
P. J. Look, Andover, 115 Main St.
R. C. Norris, Methuen, 247 Broadway.
G. L. Richardson, Haverhill, 94 Emerson St.
A. F. Shea, Lawrence, 62 Bradford St.
F. W. Snow, Newburyport, 24 Essex St., E. C., M. N. C.
T. N. Stone, Haverhill, 3 Washington Sq.
C. F. Warren, Amesbury, 1 School St.

Essex South
Bernard Appel, Lynn, 281 Ocean St., V. P.
H. A. Boyle, Middleton, Essex Sanatorium.
C. P. Brown, Swampscott, 74 Humphrey St.
Hanford Carvel, Gloucester, 1038 Washington St.
C. L. Curtis, Salem, 101 Federal St., A. M. N. C.
R. E. Foss, Peabody, 125 Main St.
S. E. Golden, Beverly, 38 Ocean St.
Loring Grimes, Swampscott, 84 Humphrey St., E. C., M. N. C.
P. P. Johnson, Beverly, 1 Monument Sq.
J. F. Jordan, Peabody, 76 Lynn St.
B. B. Mansfield, Ipswich, 4 Green St.
A. E. Parkhurst, Beverly, Monument Sq.
O. S. Pettingill, Middleton, Essex Sanatorium.
W. G. Phippen, Salem, 31 Chestnut St., Ex-Pres.

E. D. Reynolds, Danvers, 48 High St.
J. R. Shaughnessy, Salem, 24½ Winter St., Sec.
J. W. Trask, East Lynn, 90 Ocean St.
C. F. Twomey, East Lynn, 80 Ocean St.
C. A. Worthen, Lynn, 19 Park St.

FRANKLIN

A. H. Wright, Northfield, 111 Main St., V. P.
F. J. Barnard, Greenfield, 479 Main St., E. C., M. N. C.
H. L. Craft, Ashfield, Sec.
A. H. Ellis, Greenfield, 58 Federal St.
W. J. Pelletier, Turners Falls, 171 Ave. A., A. M. N. C.
H. G. Stetson, Greenfield, 39 Federal St., Ex-Pres.

HAMPDEN

G. B. Corcoran, West Springfield, 84 Park St., V. P.
F. H. Allen, Holyoke, 16 Fairfield St.
T. S. Bacon, Springfield, 69 Maple St.
E. P. Bagg, Holyoke, 207 Elm St., Vice-President.
W. C. Barnes, Springfield, 146 Chestnut St., Sec.
J. B. Bigelow, Holyoke, 109 Suffolk St.
J. M. Birnie, Springfield, 146 Chestnut St., Ex-Pres.
W. A. R. Chapin, Springfield, 121 Chestnut St.
J. L. Chereskin, Springfield, 333 Bridge St.
J. P. Derby, Springfield, 20 Maple St.
A. J. Douglas, Westfield, 93 Elm St.
E. C. Dubois, Springfield, 174 Buckingham St.
P. E. Gear, Holyoke, 188 Chestnut St.
Frederic Hagler, Springfield, 20 Maple St.
G. D. Henderson, Holyoke, 312 Maple St.
E. A. Knowlton, Holyoke, 207 Elm St., A. M. N. C.
M. W. Pearson, Ware, 19 Pleasant St.
A. G. Rice, Springfield, 33 School St., M. N. C.
G. L. Schadt, Springfield, 44 Chestnut St., President-Elect.
G. L. Steele, Springfield, 20 Maple St., E. C.

HAMPSHIRE

Mary P. Snook, Chesterfield, V. P.
A. J. Bonneville, Hatfield, 60 Main St.
W. J. Collins, Northampton, 136 Main St., M. N. C.
L. N. Durgin, Amherst, 66 Amity St., E. C., A. M. N. C.
J. R. Hobbs, Williamsburg, Main St., Sec.
L. B. Pond, Easthampton, 115 Main St.

MIDDLESEX EAST

J. M. Wilcox, Woburn, 6 Bennett St., V. P.
J. H. Blaisdell, Winchester, Office Boston, 45 Bay State Rd., A. M. N. C.
Richard Dutton, Wakefield, 33 Avon St.
E. M. Halligan, Reading, 37 Salem St.
J. H. Kerrigan, Stoneham, 481 Main St.
K. L. Maclachlan, Melrose, 1 Bellevue Ave., Sec., E. C.
G. R. Murphy, Melrose, 244 Main St.
R. W. Sheehy, Winchester, 21 Washington St.
R. R. Stratton, Melrose, 538 Lynn Fells Parkway, M. N. C., C.

MIDDLESEX NORTH

W. H. Sherman, Lowell, 9 Central St., V. P.
M. L. Alling, Lowell, 9 Central St.
H. R. Coburn, Lowell, 202 Merrimack St.
W. M. Collins, Lowell, 174 Central St., E. C., M. N. C.
R. L. Drapeau, Lowell, 310 Merrimack St., Sec.
D. J. Ellison, Lowell, 8 Merrimack St., A. M. N. C.
A. R. Gardner, Lowell, 16 Shattuck St.
W. F. Ryan, Lowell, 219 Central St.
M. A. Tighe, Lowell, 9 Central St., Secretary.

MIDDLESEX SOUTH

H. F. Day, Cambridge, Office Boston, 412 Beacon St., V. P.
C. F. Atwood, Arlington, 821 Massachusetts Ave.
E. W. Barron, Malden, Office Boston, 20 Ash St.
W. B. Bartlett, Concord, 28 Monument St.
Harris Bass, Everett, 351 Broadway.
S. M. Biddle, Cambridge, 206 Huron Ave.
E. H. Bigelow, Framingham Center, 31 Pleasant St., Ex-Pres.
G. F. H. Bowers, Newton Highlands, 156 Woodward St.
R. N. Brown, Malden, 621 Main St.
R. W. Buck, Waban, Office Boston, 5 Bay State Rd.
E. J. Butler, Cambridge, 25 Garden St.
B. F. Conley, Malden, 51 Main St.
P. A. Consales, Belmont, Office Boston, 481 Beacon St.
D. F. Cummings, Natick, 12 East Central St.
C. H. Dalton, Somerville, 440 Broadway.
C. L. Derick, Newton Highlands, Office Boston, 412 Beacon St.
J. E. Dodd, Framingham, 259 Union Ave.
J. G. Downing, Newton, Office Boston, 520 Commonwealth Ave.
A. W. Dudley, Cambridge, 1740 Massachusetts Ave.
C. W. Finnerty, West Somerville, 5 Pearson Rd.
E. R. Fleming, Medford, 322 Boston Ave.
H. Q. Gallupe, Waltham, 751 Main St.
F. W. Gay, Malden, 105 Salem St.
H. G. Giddings, Newton Centre, Office Boston, 270 Commonwealth Ave.
H. W. Godfrey, Auburndale, 14 Hancock St.
B. I. Goldberg, Newton Centre, Office Boston, 481 Beacon St.
A. D. Guthrie, Medford, 408 Salem St.
A. M. Jackson, Everett, 512 Broadway.
E. E. Kattwinkel, West Newton, 65 Sterling St.
A. A. Levi, Newton, Office Boston, 481 Beacon St., Sec.
F. P. Lowry, Newton, 313 Washington St.
A. N. Makechnie, Cambridge, 14 Upland Rd.
R. A. McCarty, Waltham, 751 Main St.
J. C. Merriam, Framingham, 198 Union Ave., A. M. N. C.
Dudley Merrill, Cambridge, 51 Brattle St.
C. E. Mongan, Somerville, 24 Central St., Ex-Pres.
J. P. Nelligan, Cambridge, 2336 Massachusetts Ave.
Dwight O'Hara, Waltham, Office Boston, 5 Bay State Rd., E. C., M. N. C.
L. S. Pilcher, Newton Centre, 43 Parker St.
Max Ritvo, Newton, Office Boston, 485 Commonwealth Ave.
E. H. Robbins, Somerville, 334 Broadway.
E. S. A. Robinson, Newton Centre, Office Jamaica Plain, 375 South St.
W. D. Roche, Marlboro, 196 Main St.
E. F. Ryan, Maynard, 74 Main St.
M. J. Schlesinger, Newton, Office Boston, 330 Brookline Ave.
W. N. Secord, Watertown, 128 Mt. Auburn St.
J. W. Sever, Cambridge, Office Boston, 321 Dartmouth St.
E. F. Sewall, Somerville, 380 Broadway.
E. W. Small, Belmont, 68 Leonard St.
H. P. Stevens, Cambridge, 1 Craigie St.
R. A. Taylor, Waltham, 550 Moody St.
H. W. Thayer, Newtonville, 355 Walnut St.

J. H. Townsend, Belmont, Office Boston, 319 Longwood Ave.
J. E. Vance, Natick, Office Boston, 510 Commonwealth Ave.
R. H. Wells, Lexington, 1430 Massachusetts Ave.
Alfred Worcester, Waltham, 314 Bacon St., Ex-Pres.
Hovhannes Zovickian, Watertown, 528 Mt. Auburn St.

NORFOLK

S. A. Robins, Roxbury, Office Boston, 636 Beacon St., V. P.
F. J. Bailey, Dorchester, 40 Hancock St.
J. R. Barry, West Roxbury, 1857 Centre St.
Carl Bearse, Boston, 483 Beacon St., E. C.
M. I. Berman, Dorchester, 1071A Blue Hill Ave.
G. F. Blood, Roslindale, 20 Belgrade Ave.
F. S. Cruickshank, Brookline, 1247 Beacon St.
William Dameshek, Brookline, Office Boston, 371 Commonwealth Ave.
F. P. Denny, Brookline, 111 High St., C.
J. J. Elliott, Roslindale, 4258 Washington St.
H. M. Emmons, Needham, Office Boston, 354 Commonwealth Ave.
J. E. Fish, Canton, Massachusetts Hospital School.
J. C. V. Fisher, West Roxbury, Office Boston, 510 Commonwealth Ave.
Susannah Friedman, Roxbury, Office Boston, 485 Commonwealth Ave.
David Glunts, Roxbury, 2 Franklin Garden.
B. T. Guild, Dorchester, Office Boston, 475 Commonwealth Ave.
D. L. Halbersleben, Brookline, Office Roslindale, 818 South St.
J. B. Hall, Roxbury, 60 Windsor St.
R. J. Heffernan, Jamaica Plain, Office Brookline, 1101 Beacon St.
Morris Ingall, Roxbury, Office Boston, 485 Commonwealth Ave.
H. J. Inglis, Chestnut Hill, Office Boston, 43 Bay State Rd., A. M. N. C.
I. R. Jankelson, Jamaica Plain, Office Boston, 483 Beacon St.
H. L. Johnson, West Roxbury, Office Boston, 520 Commonwealth Ave.
C. J. Kickham, Brookline, Office Boston, 524 Commonwealth Ave.
C. J. E. Kickham, Jamaica Plain, Office Boston, 12 Bay State Rd.
E. L. Kickham, Brookline, Office Boston, 270 Commonwealth Ave.
D. L. Lionberger, Dedham, Office Roslindale, 3 Conway St.
D. S. Luce, Canton, 553 Washington St.
C. M. Lydon, Dorchester, 276 Bowdoin St.
D. L. Lynch, Roslindale, Office Boston, 245 State St.
T. F. P. Lyons, Milton, Office Boston, 270 Commonwealth Ave., Sec.
Charles Malone, Jamaica Plain, 46 St. John St.
F. P. McCarthy, Milton, Office Boston, 371 Commonwealth Ave.
F. J. Moran, Dedham, 395 Washington St.
M. W. O'Connell, West Roxbury, Office Boston, Boston City Hospital.
H. C. Petterson, West Roxbury, Office Boston, 29 Bay State Rd.
Frederick Reis, Jamaica Plain, Office Boston, 416 Huntington Ave.
S. M. Saltz, Roxbury, 294 Seaver St.
D. D. Scannell, Jamaica Plain, Office Boston, 475 Commonwealth Ave., M. N. C.
J. A. Seth, Milton, Office Boston, 47 Bay State Rd.
Nathan Sidel, Brookline, Office Boston, 483 Beacon St.
J. W. Spellman, Milton, Office Brookline, 1101 Beacon St.
M. H. Spellman, Jamaica Plain, Office Boston, 475 Commonwealth Ave.
W. J. Sullivan, Dorchester, 1993 Dorchester Ave.
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MEDICAL PROGRESS

PHYSIOLOGY*

HEBBEL E. HOFF, M.D.†
NEW HAVEN, CONNECTICUT

THE increasing threat of war has directed the attention of physicians to some of the immediate medical problems created by war. The background of many of these problems lies in the work done in peacetime, with entirely different objectives. With this in mind, I have devoted this year's report to a consideration of the physiologic background of certain questions of topical interest.

ANOXEMIA IN THE NERVOUS SYSTEM

The brain is unusually susceptible to deprivation of oxygen. In contrast to many other tissues such as muscle, the dominant respiratory process in the brain in vivo is the oxidation of glucose, so that even momentary lack of oxygen seriously interrupts its metabolic activity.[1] Complete interruption of cerebral circulation produces unconsciousness in six to eight seconds, and all electrical signs of activity in the brain are obliterated in twenty seconds.[2] An anemia of two to fifteen minutes gives rise to irreversible changes in the cortex and medulla, and temporary blindness may follow anoxemia for five minutes.[3] The centers in the brain stem regulating respiration and the cardiovascular system are much more resistant, and may show recovery after complete anemia for as long as thirty to fifty minutes. This difference in the vitality of respiratory and cardiovascular centers and the cortex probably accounts for the cases not infrequently seen in which patients recover from respiratory failure under anesthesia, or from carbon monoxide poisoning, but show signs of irreversible cortical damage.

The electrical activity of the brain may be used as an index of cortical anemia.[4] Usually, no changes are seen until the venous blood from the brain shows an oxygen content below 30 per cent saturation (normal, 60 per cent). Then the faster alpha waves begin to diminish in frequency and amplitude, after an occasional initial period of increased rate. There then appears an augmentation in frequency and amplitude of the slower delta waves, and if the anemia persists and deepens, these waves may entirely replace the alpha waves.[5] Seizurelike discharges may occur.[4, 5] Finally, all waves disappear.

States in which there is a cerebral anemia result in a similar type of electroencephalogram, characterized by the predominance of delta waves. Among these are carbon monoxide poisoning, nitrous oxide anesthesia with insufficient oxygen,[6] a reduced blood carbon dioxide level, and increased intracranial pressure.[4] It appears that a low blood carbon dioxide level causes constriction of the cerebral arteries, which may be sufficient to produce anemia. Increased intracranial pressure, whether from tumor, trauma or hemorrhage, apparently produces no changes until the intracranial pressure approaches to within 40 mm. of mercury of the arterial blood pressure.[7, 8] Any increase from this point diminishes the cerebral circulation, and is followed by the characteristic signs of cerebral anoxemia.

Many authors have emphasized the close relation between the influence of anoxemia and hypoglycemia on the brain.[1, 5] The general symptoms as seen in the various so-called "shock" treatments of mental illnesses appear to be much the same, whether the shock is produced by low blood sugar or by anoxemia.[4] The electroencephalogram in hypoglycemia is also very similar to that in anoxemia.[9–11] Changes appear when the blood-sugar level (in the internal jugular veins) falls below 35 mg. per 100 cc., and consist in an early decrease in rate and amplitude of alpha waves, followed by an increase in amplitude and frequency of the delta waves.[4] When the dependence of the brain on glucose oxidation is recalled, it is understandable that deprivation of either glucose or oxygen should have similar effects.[1]

These studies have an obvious relation to the problems of aviation. As the altitude at which a plane flies increases, the partial pressure of oxygen decreases, and symptoms of anoxemia begin to appear. In general, most persons begin to show increased respiration at from 6000 to 10,000 feet. Symptoms of sleepiness, fatigue and headache usually appear at 10,000 to 12,000 feet. Impairment of cerebral functions may be evident at from 8000 to 15,000 feet, individual variations being considerable. Above 20,000 feet, there is a critical danger to life, and coma and convulsions may be

All articles in this series will be published in book form; the current volume is *Medical Progress: An annual, 1940* (Springfield, Illinois: Charles C Thomas Company, 1941. $4.00).

*From the Laboratory of Physiology, Yale University School of Medicine.
†Associate professor of physiology, Yale University School of Medicine.

expected. Naturally, physical exercise or small amounts of carbon monoxide in the inspired air may greatly lower the altitude at which alarming effects of anoxemia may occur.[12]

Recent studies of the reaction of respiratory mechanism to anoxemia are of interest because of their applicability to the problems of high-altitude flying and of general anesthesia. Schmidt and Comroe have for some time called attention to the major importance of the carotid body in the mediation of respiratory changes in response to low oxygen, whereas the respiratory center itself responds to increased carbon dioxide. In passing, it should be remarked that one of these authors[13] has recently made series of minute injections into the respiratory center of acid solutions or of solutions containing sodium bicarbonate and carbon dioxide. Although the former solutions had very little influence on respiration, the latter produced a prompt increase in respiration. These experiments, therefore, add to the evidence that carbon dioxide, and not pH, is the adequate stimulus to the respiratory center.

Schmidt and Comroe[12] have offered evidence indicating that during anoxemia, or during deep narcosis, the respiratory center may be seriously impaired, so that the main drive to the respiration is afforded by the carotid body. When, in such circumstances, the oxygen content of the blood is suddenly increased, the carotid body also ceases to function, and the center is unable to carry out its function alone. Respiration then ceases, and may fail long enough to cause death. The authors suggest that this may be the mechanism of respiratory failure in operations in which oxygen is administered after a period of anoxemia, and warn that delay in starting to use oxygen may create an anoxemia that will make possible similar accidents when oxygen is finally taken.

The autonomic nervous system is also involved in the reactions to anoxemia. The increased rate of the heart, the elevation of the blood pressure and the contraction of the spleen appear early and become increasingly noticeable as the anoxemia progresses. They are seen in experimental animals and in man during exposure to low atmospheric pressures,[14] and point to stimulation of the sympathetic nervous system. There may often occur an abrupt crisis at a degree of anoxemia near that which may produce unconsciousness; the heart suddenly slows, auriculoventricular block appears, and the subject becomes pale and sweaty, and faints. These symptoms point to an overaction of the parasympathetic nervous system and, thus, may in part be prevented by atropine.

Such observations do not permit the conclusion that the type of autonomic response depends on the intensity of the anoxemia. On the contrary, there is recent evidence that during asphyxia both sympathetic and parasympathetic systems are stimulated simultaneously (Hodes,[15] Feldman, Cortell and Gellhorn[16]). Hodes compared the response of normal and sympathectomized animals to exercise and found that, although the normal cat showed a marked acceleration, the sympathectomized cat showed a decrease in rate. He suggests that muscular exercise produces a local vasodilatation, which cannot be compensated for in the sympathectomized cat. The blood pressure then falls, cerebral circulation becomes inadequate, and cerebral anoxemia results. The cerebral anemia then stimulates the sympathetic and parasympathetic centers, and the sympathectomy permits the parasympathetic responses to develop without opposition. Feldman, Cortell and Gellhorn arrive at the same conclusion after demonstrating that, in the normal rat, anoxemia (breathing 7 per cent oxygen) causes a rise in blood sugar, whereas it causes a hypoglycemia in the adrenalectomized animal.

Anoxemia in the Heart

The electrocardiogram serves as the most valuable indication of the presence and extent of anoxemia in the heart. Analysis of the voluminous literature of clinical and experimental studies permits the following general conclusions to be drawn. When the oxygen content of arterial blood is reduced from 30 to 50 per cent by any of a variety of procedures, such as breathing air containing a reduced (12 per cent) oxygen content, a progressive diminution of the height of the T wave is noted. This reduction in the amplitude of the T wave, which may eventually progress to inversion, is seen in all three standard leads, and has been observed in man as well as experimental animals.[17–19] It has been observed in aviators during ascents to 5000 feet or over without supplementary oxygen.[20] There is occasionally some displacement of the RST segment, usually downward. When the oxygen content of the arterial blood is reduced below 50 per cent saturation, changes resembling those in myocardial infarction appear. These include further displacement of the RST segment (oftener elevation), and the development of the so-called "coronary T wave." Increase in the amplitude of the Q wave is not a prominent feature. If the deficiency of oxygen is neither too severe nor too prolonged, no anatomic changes are found in the heart, although the electrocardiographic changes may persist for some time.[21] If the anoxemia is severer, and persists but does not entail complete deprivation of oxygen, some necrosis will occur, which is distributed in small

foci that are largely subendocardial. These will be replaced in time by diffuse fibrous tissue. Carbon monoxide poisoning in man has been found to produce just such a picture.[22] Various German authors have for some time pointed out that this type of necrosis occurs in coronary disease in which the vessels are narrowed enough to cause serious restriction of the coronary blood supply but still permit sufficient circulation to prevent massive infarction.

Master and his collaborators[23] have recently shown that this type of myocardial damage is associated with electrocardiographic changes indicative of the lesser degree of anoxemia described above, that is, reduction in amplitude of the T wave and slight depression of the RST segment, and propose the term "coronary insufficiency" to designate the condition. Their use of the term "coronary occlusion" for the state associated with massive myocardial infarction is perhaps less justified, especially in view of the most recent work of Blumgart, Schlesinger and Zoll,[24] who show that coronary occlusion may often occur without infarction.

The Nature of the Electrocardiogram

An adequate explanation for the changes seen in the electrocardiogram during anoxemia, and after massive infarction of the myocardium, is not yet available, largely because there is as yet no adequate explanation for the normal electrocardiogram. There has been, however, some recent work on this subject. Some time ago, Eppinger and Rothberger[25] made superficial lesions in the heart by various methods (for example, the injection of corrosives) and found that the surface injuries produced marked effects, whereas deep injuries, not affecting the surface, evoked only minor changes, if any. They concluded that the electrocardiogram was largely determined by the electrical activity of the surface of the heart. Recently the subject has been reinvestigated by other methods,[26, 27] which confirm the thesis of Eppinger and Rothberger.

One of the procedures[27] employed in the above studies consisted in treating the heart with isotonic solutions of potassium chloride, which were found to cause a local and reversible depolarization, similar to that produced by an injury. Areas of the surface of the ventricles thus treated failed to develop their normal action potentials, and the influence of this failure on the electrocardiogram could be determined. When the left ventricle was so treated, elevation of the RST segment appeared in all three conventional leads. The elevation increased as more and more of the surface was involved, until finally an upright monophasiclike complex remained. Since this remained after maximal injury to the left ventricle, it was concluded that this complex represented the contribution of the right ventricle to the electrocardiogram, that is, the "dextrocardiogram." Injury to the right ventricle, on the contrary, produced a depression of the RST segment, which increased with extension of the area of injury until an inverted monophasiclike complex remained after maximal injury to the right ventricle. It appeared that this complex represented the contribution of the left ventricle to the electrocardiogram, that is, the "levocardiogram." Other evidence proved that the dextrocardiogram and levocardiogram were in fact derived from the right and left ventricles, respectively. When these curves were plotted, and summed algebraically, complexes closely resembling the normal QRST complexes were obtained. It was concluded that the normal electrocardiogram results from the algebraic summation of the dextrocardiogram and levocardiogram.

Elevation of the RST segment may therefore be taken to indicate damage to the left ventricle, whereas depression denotes damage to the right ventricle. When the lesion is restricted to a single ventricle, the RST segment is displaced in the same direction in all three conventional leads, although the greatest displacement occurs in Lead 2. When, however, the injury involves areas of both the right and the left ventricle, Leads 1 and 3 show the maximal changes, and the displacement in one lead is opposite to that in the other. With lesions of the anterior surface of the right and left ventricles, elevation of RST occurs in Lead 1, together with depression in Lead 3. Posterior damage involving both ventricles causes depression of RST in Lead 1 and elevation in Lead 3.[28]

According to the theory outlined above, the T wave represents the algebraic summation of the terminal portions of the dextrocardiogram and levocardiogram. Normally, the right ventricle is excited slightly before the left, producing the normal upright R wave. If the durations of the dextrocardiogram and levocardiogram are equal, the latter would be expected to terminate after the former, since it begins somewhat later. The T wave would therefore be downward. If for any reason the dextrocardiogram were prolonged beyond the termination of the levocardiogram, the T wave would be upright. Alterations in the relative durations of the dextrocardiogram and levocardiogram were actually produced by local heating and cooling of the surface of the ventricles, and concomitant changes in the T wave of the electrocardiogram appeared that were in every detail consistent with these theoretical postulations.[29]

Studying the effects of coronary occlusion by means of direct and semidirect leads, Jochim and his associates[30] concluded that three different factors play a part in the production of the characteristic electrocardiographic patterns: disturbances in conduction, partial or complete depolarization in the area of greatest injury, with consequent failure of action potentials from this area, and delayed repolarization in tissue that although damaged remains excitable and produces an action potential when stimulated. In view of the similarity between the monophasic action current and the dextrocardiogram and levocardiogram, it is more than likely that the postulates of these authors, which now apply to the electrogram derived from direct leads, may be extended to the electrocardiogram. The effects of anoxemia on the electrocardiogram should then find explanation in the influence of anoxemia on the dextrocardiogram or levocardiogram.

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Shock

Freeman et al.[31] believe that at least one factor in the causation of shock is overactivity of the sympathetic nervous system. It may be induced reflexly by the fall in blood pressure in hemorrhage, by the afferent nervous stimulation in trauma or by the cortical stimulation of concussion, and it serves the normal function of maintaining blood pressure. If, however, the stimulus that has produced the overactivity continues, the latter may progress to a pathologic degree. Peripheral arterial constriction will then diminish blood flow in the periphery to such a degree that capillary anoxemia occurs. Capillary anoxemia will be followed by dilatation of the capillaries, by fluid loss from the blood stream, by increased blood concentration, and by a progressively falling blood pressure — in other words, by shock. Freeman has shown that shock may develop in animals following slow constant infusion of adrenalin into the blood stream, which is confirmed by Swingle et al.[32] Other authors doubt whether the adrenalin-sympathetic theory can be applied to all types of shock,[33] and present experiments which appear to show that the administration of adrenalin may be a helpful therapeutic measure in shock consequent to intestinal manipulation.[34]

It is more than likely that a variety of factors are involved in the genesis of the syndrome generally recognized as shock. One important example comes from renewed interest in the influence on shock of extracts of the adrenal cortex and allied substances. Swingle and his co-workers[32] have demonstrated that additional cortical extract will protect against shock caused by trauma, by intraperitoneal injections of glucose or by injections of adrenalin in adrenalectomized animals maintained on adequate amounts of cortical extract. These authors then studied the effectiveness in these situations of desoxycorticosterone, a synthetic substance that appears to have the same effect on mineral metabolism as cortical extract. Desoxycorticosterone, used prophylactically, protected against shock induced by trauma, by injection of glucose into the peritoneal cavity and by injection of adrenalin. It had no effect on the shock following intestinal stripping.

The efficacy of adrenocortical extracts in the prophylaxis of shock of course raises the question of the relation of potassium to the various symptoms of shock. There is evidence that serum potassium may rise in shock.[35] Study of the influence of slowly rising concentrations of potassium in normal dogs makes it questionable whether increases in potassium, below levels required to kill the animal, cause any of the symptoms of shock.[36] Whether potassium is responsible for death in shock cannot be stated. Recent studies have established criteria by which the question may be judged.[37] Serum potassium should be in the neighborhood of 15 milliequiv. per liter in the presence of a normal serum calcium. Much higher levels of potassium may be reached if the calcium is elevated.[38] Accompanying the rise in serum potassium, a characteristic sequence of electrocardiographic changes should appear in the following order: increase in the amplitude of the T wave, depression of the ST segment, disappearance of the P wave, intraventricular block and arrest, or terminal ventricular fibrillation. At present the only situation (apart from poisoning from injection of potassium) in which accumulation of potassium has been shown to have caused death is in experimental anuria.[39] In dogs in which anuria was induced by ligation of the ureters or by removal of the kidneys, potassium steadily accumulated in the blood, and when it reached toxic levels the animals died.

There is now at hand a report of fatal shock in man accompanied by anuria, following a bomb blast.[40] Potassium in the serum rose to about 11 milliequiv. per liter at death. The patient died suddenly, with a rapid fall in blood pressure and an irregular pulse. The evidence in this case is entirely consistent with death from potassium poisoning, but without serial electrocardiograms it is difficult to be certain that this was actually so. It appears that the combination of shock and anuria will be a not infrequent occurrence after bomb-blast injuries.[41]

The use of desoxycorticosterone to prevent shock

may not be entirely without dangers. Extreme muscular weakness resembling familial periodic paralysis has followed the repeated injection in dogs of large doses of the substance.[42] There are also reports[43, 44] of the development of cardiac failure in man when large doses are given, and the condition also occurs in dogs. The difficulty may be diagnosed by the low serum potassium, and potassium added by mouth seems to be a specific remedy. It is possible that under some conditions prolonged periods of low potassium may result in pathologic changes in the heart.[45]

Histamine

The subject of shock naturally recalls the question of the role of histamine. Two general reviews of the subject are most valuable, because in both an attempt is made to delimit the action of histamine, and to indicate the circumstances in which histamine is not the causative agent, as well as those in which it is implicated.[46, 47] Evidence for histamine release is perhaps best established in the "triple response" of the skin to injury (Lewis[48]). The resemblance of the triple response to the response of local anaphylaxis in the skin led Lewis to conclude that the antibody-antigen reaction in sensitized skin cells represented a special form of cell injury, which like other stimuli injurious to cells caused the release of a preformed histaminelike substance. This naturally led to the concept that many responses in anaphylaxis of other types are due to the liberation of histamine.

The release of histamine seems to be particularly well established in the physical and nervous allergies. The allergic reaction to cold or heat, which may be accompanied by extreme vasodilatation and a fall in blood pressure, also causes an increased secretion of highly acid gastric juice, which appears to be good evidence that histamine is involved. The urticarial reactions to nervous stimuli, exercise and temperature changes reported by Grant, Pearson and Comeau[49] are similar to reactions caused by histamine, and are presumably due to the release of histamine by cholinergic nerve fibers in the skin of susceptible persons. In anaphylactic shock in dogs and rabbits, release of histamine can account for the major symptoms. Evidence is available to implicate histamine as the active factor in other types of human allergy, but cannot be said to be conclusive. The demonstration that histamine can be liberated in the skin of susceptible persons by cholinergic nervous stimulation strengthens the possibility that asthma of nervous origin may arise from the release of histamine in the lungs by vagal stimulation.

Histamine may also play a part in the genesis of the symptom complex arising from bacterial or other intoxication. The acute collapse following administration of the toxin of *Staphylococcus aureus* has been shown to result from the liberation of histamine.[50] Certain snake venoms also release histamine. In all these circumstances, other effects of the toxic agent may also appear, and may produce symptoms. Thus mercuric chloride liberates histamine,[50] but it also coagulates protein, both factors being important. On the whole, Feldberg[46] concludes that the acute effects of the intoxication are likely to be histamine effects, whereas the chronic effects are likely to be results of other actions of the intoxicating agent. It is of interest that a disease in horses can be shown to be due to the action of certain bacteria in the gastrointestinal tract, which decarboxylates the aminoacid histidine to form histamine. Feldberg also points out that a diversity of quite unrelated factors may operate to liberate histamine, such as simple coagulation of cell protein, dissolution of lipoprotein combinations within the cell and mechanical injury. The substance therefore appears to be in some sort of union with the cellular protein.

Some reports have appeared of the successful use of histaminase to reduce the response of reactive cells to histamine. Other attempts have not been uniformly successful.[46]

High-Protein Diet as a Protection Against Liver Injury

The well-established practice of giving diets high in carbohydrate in conditions in which liver damage is a factor may require modification in view of recent experimental data, which indicate the even greater protection afforded by a diet high in protein.[51] Using chloroform as the toxic agent, Ravdin and his associates[52] found that starved animals were more susceptible, whereas animals fed high-protein diets were much more resistant than normal animals. Miller and Whipple[53] have confirmed these experiments in the dog. They find that a normal well-fed dog will tolerate an hour of chloroform anesthesia without showing liver necrosis. Normal dogs fasted for three days respond to the same treatment by the development of marked hyaline central necrosis of the liver, marked lowering of prothrombin and fibrinogen in the blood, increase in the bile pigment in the liver, increase in urinary nitrogen and clinical signs of intoxication. Some animals die in two to four days. Dogs with low serum protein (low-protein diets or plasmapheresis) can hardly tolerate fifteen to twenty minutes of anesthesia. A single large protein feeding will markedly increase the resist-

ance of these dogs to chloroform. The protective effect of certain irritant substances (sodium ricinoleate and colloidal carbon) may be due to the inflammatory reactions they evoke, and to the consequent increased protein catabolism.[54] There is some evidence that the sulfur-containing aminoacids have a role in the protective influence of protein.

A variety of other toxic agents affecting the liver are also shown to be influenced by a high-protein diet. The most important is perhaps arsphenamine, which at times may cause liver damage when used therapeutically. Using admittedly high doses (0.03 gm. per kilogram) Messinger and Hawkins[55] found that both high-protein and high-carbohydrate diets were protective, whereas fat diets were harmful. The toxic effects of selenium on the liver are also determined by the diet. A high-protein diet greatly reduces the liver damage produced by feeding selenium-containing wheat.[56] Lewis, Schultz, and Gortner,[57] who noted the increased resistance to selenium afforded by a diet containing 30 per cent casein rather than 6 per cent, found that methionine was protective, whereas cystine in equivalent dosage was not, although both are sulfur-containing aminoacids.

These studies emphasize two very practical points. First, they call attention, if this is still necessary, to the extreme hazards accompanying the use of chloroform as an anesthetic, especially if anoxemia is also a factor.[58] Secondly, they suggest that a high-protein, high-carbohydrate diet is required in any condition in which liver damage must be combated or prevented.

Associated with this problem are reports showing that a gum acacia, given for its osmotic-pressure effect, accumulates in the liver and depresses certain liver functions.[59, 60] Large quantities of the material may accumulate in the liver, and there act to diminish the production of plasma proteins, affecting most seriously the fibrinogen.[61, 62] Other liver-damaging substances enhance the depression of plasma proteins by acacia.[63] The depression of plasma proteins may last for several months after cessation of acacia injections, and the enlarged acacia-filled liver may persist for a long time. There is one report of its presence in the liver of a child to whom it was given six years previously.[64] Acacia does not appear to affect the production of bile salts or bile pigments, nor is it responsible for the alteration in blood and bile cholesterol.[65]

333 Cedar Street

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27271

PRESENTATION OF CASE

First Admission. A forty-four-year-old Italian carpenter entered with the complaint of intermittent pain in the epigastrium of about three years' duration. Starting as a sensation of "knotting" in the epigastrium frequently associated with "burning" between the shoulder blades, it became progressively severer, developing into a sharp pain, which characteristically occurred thrice daily at 11 a.m., 5 p.m. and 11 p.m. and was readily relieved for a few hours by food or powders. Occasionally, the patient was awakened from sleep by an attack of pain, and on such occasions he usually felt nauseated. He rarely vomited, and the vomitus was never blood stained or black. He had never noted any black stools. During the first two years, his attacks had been interspersed between comparatively long periods of complete freedom from symptoms. In the last five months there had been no significant remission. He had lost about 25 pounds in the year before entry.

Nine months before admission the patient had visited the Out Patient Department, where a gastrointestinal series was reported as follows: "Examination of the esophagus shows no evidence of varices or herniation. The stomach is high in position and fills well. There is definite tenderness over the lesser curvature about 6 cm. from the hiatus, where there is an ulceration 1.5 cm. in width which extends 1 cm. outward from the body of the stomach. The antrum shows considerable spasm. The duodenal cap fills well and shows normal relief. The findings are those of a large gastric ulcer on the lesser curvature." A six-meal bland diet was prescribed, with transient relief, but was not adhered to. An x-ray examination after seven months still showed the ulcer crater, although it seemed definitely shallower than on the first examination. Because of his unsatisfactory progress, hospitalization was advised.

The past history recorded an episode of "anemia" during service in the Italian army twenty years before, which was treated with a series of thirty-six daily gluteal injections. The patient's father was living and well at seventy-two, his mother had died at fifty of unknown cause. One brother had attacks of "stomach trouble" associated with the vomiting of bright-red blood. His wife had had four children, all of whom were living and well.

Physical examination showed a sparse, muscular man lying comfortably flat in bed in no distress. The skin and mucous membranes showed no icterus or pallor. The nasal septum was deviated to the left. The teeth were carious, with some bleeding of the gums. The heart and lungs were normal, the blood pressure 100 systolic, 70 diastolic. Abdominal examination disclosed no tenderness, and no masses or organs could be felt. Rectal examination showed small hemorrhoidal varices. Reflexes were normal.

The temperature was normal, the pulse from 70 to 80, and the respirations from 15 to 20.

Laboratory examinations included a negative blood Hinton reaction. The red-cell blood counts ranged from 4,700,000 to 4,900,000, the hemoglobins from 84 to 90 per cent (Tallqvist), and the white-cell counts from 7000 to 11,000. The differential count was 54 per cent polymorphonuclears, 34 per cent lymphocytes, 21 per cent monocytes and 2 per cent eosinophils. There were no abnormal cells, and the red cells showed no abnormalities. The urine varied from 1.016 to 1.030 in specific gravity and never showed albumin, sugar, diacetic acid or bile. The sediment was normal. Fifteen stools were brown in color and negative to the guaiac test. Two serum-protein determinations showed 6.6 and 7.0 gm. per 100 cc. The nonprotein nitrogen was 22 mg. per 100 cc., and the blood chloride 101.6 milliequiv. per liter. A gastric analysis showed no free hydrochloric acid in the fasting contents, but 50 units after histamine.

The patient was placed on a regime of strict bed rest, a first-stage ulcer diet, Amphogel and tincture of belladonna supplemented by subcutaneous atropine. His improvement was steady. Three x-ray examinations at intervals of approximately two weeks reported respectively: "The ulcer crater is definitely smaller and now measures 8 mm. in diameter"; "The crater is much smaller and now measures approximately 2 to 3 mm. in diameter"; "An ulcer crater is no longer visible." All reports, however, including the last, described shortening of the lesser curvature and spasm in the prepyloric area. Deformity of the duodenal cap was mentioned twice, but no spasm or evidence of active ulceration was noted. Two gastroscopic examinations were performed during the same period. The first described a typical benign ulcer 0.5 cm. in diameter, with slight granularity of the

surrounding mucosa. On the second examination no ulcer could be seen. The patient was discharged after nine weeks, apparently relieved.

Second Admission (nine months later). During the succeeding period, the patient was seen at irregular intervals in the gastrointestinal clinic of the Out Patient Department. Despite adherence to a six-meal diet, he had some discomfort almost every day, usually a sensation of burning between the shoulder blades rather than epigastric pain. X-ray examinations one and two months after discharge were inconclusive, mentioning spasm in the prepyloric area and questionable irregularity of the lesser curvature proximal to the area of spasm. The following month a gastroscopic examination demonstrated recurrence of the ulcer, with a crater 1 cm. in diameter and approximately 3 mm. in depth. The patient was advised to re-enter the hospital, but refused and disappeared for an interval of six months. On return he reported an episode two months before of increasing pain for several days followed by vomiting a large amount of bright-red blood and fainting. He was taken to another hospital, where he remained one week. In the two weeks before entry his pain had become severer and was no longer relieved by food or soda. An x-ray examination in the Out Patient Department was reported as follows: "Examination shows a normal esophagus and a high stomach, which lies in the transverse position and is beyond the reach of palpation." An ulcer could again be demonstrated on the lesser curvature almost as large as that noted at the first examination. The lesser curvature in the neighborhood of this ulcer appeared somewhat rigid and edematous. There was again spasticity of the prepyloric area, but this area opened up during the examination, filled and emptied well. The duodenal cap and remainder of the duodenum showed nothing abnormal. The mucosal folds in the stomach appeared somewhat hypertrophic. The diagnosis was "ulcer of the lesser curvature, which appears benign." Hospitalization was again advised, and the patient re-entered.

Physical examination was essentially unchanged, except that the mucous membranes showed pallor and the blood pressure was 140 systolic, 90 diastolic. His weight was 131 pounds, as compared with 126 on the first admission. The heart, lungs and abdomen were entirely normal. Examination of the blood showed a red-cell count of 4,900,000 with a hemoglobin of 10.5 gm., and a white-cell count of 8800. The differential count was 57 per cent polymorphonuclears, 24 per cent lymphocytes, 8 per cent monocytes, 10 per cent eosinophils and 1 per cent basophils. There were moderate achromia and variation in the size and shape of the red cells. One of two urine examinations showed a + test for albumin. Two stool examinations were guaiac negative. A gastric analysis showed 18 units of free hydrochloric acid in the fasting contents and 41 units after histamine. The material was guaiac negative.

On the sixth hospital day a laparotomy was performed.

Differential Diagnosis

Dr. Helen Pittman: This seems to me like a very run-of-the-mill case.

I do not know anything about Italian medicine, but according to the little I know about French medicine everything is treated by injection, and I should assume that this patient had just what the record states, "an episode of anemia," and I am going to disregard it as having anything to do with the rest of the case.

The only interesting points in the laboratory findings are the 21 per cent monocytes, not borne out by a later count, and the absence of free acid in the fasting contents but the presence of acid after histamine. The two differential counts do not compare. One shows 21 per cent monocytes and 2 per cent eosinophils, and the next had 8 per cent monocytes and 10 per cent eosinophils. I shall assume that it is not very important.

Perhaps this would be a good time to see the x-ray films.

Dr. Tracy B. Mallory: I shall show lantern slides of the films. The lesion is so obvious that I can point it out without benefit of a roentgenologist. This was taken in March, 1939, showing a good-sized crater, situated just about the middle of the lesser curvature. The second slide shows the stomach a year and a month later, and the crater is absent. In the third picture, one year subsequently, it is back again and is of the same size as in the first examination.

Dr. Pittman: I think that this is a perfectly classic story of recurrent gastric ulcer, which did very well in the hospital under supervision. When the patient returned home and was not under supervision it recurred, although he said he had adhered to his diet. He was operated on because of the danger of its being carcinomatous. I think he should have been operated on, and I should think no one would be able to tell what they were going to find.

The physical findings on the whole were quite normal. The patient developed no anemia; he had one period of frank bleeding, but no occult blood. His weight kept up; he had always had acid after histamine, and free acid was present at the second entry. I do not believe it is possible to

say whether he had a carcinoma or whether he had an ulcer. On the basis of the facts that he had not developed anemia, that he had not lost weight and that the general condition apparently was very good, I am going to guess that when they operated he still had a benign gastric ulcer.

Dr. Aubrey O. Hampton: We could not tell any more than anyone else about this case. The diagnosis depends on the histology. The fact that the ulcer reduced in size — perhaps it did not quite disappear, but we thought it did — does not rule out carcinoma. Although some years ago I should have been fairly certain it was a benign ulcer, at present I am skeptical.

Dr. Edward B. Benedict: I did three gastroscopies on this patient. At the first examination the lesion looked essentially benign. I thought he should be carefully followed. Two weeks later I did another gastroscopy and saw no ulcer, but pointed out the fact that it might be hiding behind the angulus. There is a blind spot where it is possible to miss an ulcer, and I still advised following him carefully by x-ray study and by gastroscopy. Two months later I saw him again and gastroscoped him, and the ulcer was there again. The recurrent ulcer still looked grossly benign, but I questioned the possibility of malignancy and advised him to enter the house. I think that from then on the history is misleading. I am quite sure he was completely lost track of for seven or eight months and not seen in the clinic. He refused to re-enter the hospital. When I next saw him this winter, x-ray study showed a definite recurrence, and the patient was advised to come in for resection.

Dr. Leland S. McKittrick: Were you or anyone else worried about the shortening of the lesser curvature described?

Dr. Hampton: No. At one time that was thought to be commoner in benign than in malignant lesions, but I do not believe it is worth anything in the differential diagnosis. It means only spasm of the longitudinal muscles. It could occur in either type of lesion.

Dr. Horatio Rogers: I should think the assumption would have been that this was malignant. With his age, with the change of symptoms and with the recurrence of the ulcer, I should have considered it malignant rather than benign. I am surprised that they assumed it the other way. What was the objection to operation at the first admission?

Dr. Benedict: We advised hospitalization on the basis of potential malignancy when first seen in the Gastrointestinal Clinic, but the patient refused to come in.

Dr. Rogers: What is the prognosis of a man of this age with an ulcer more than a centimeter in diameter in the stomach?

Dr. Hampton: I do not know.

Dr. McKittrick: Do you not believe, if you want to be critical, you should go back nine months and ask why he had not been hospitalized when the diagnosis of gastric ulcer was first made?

Dr. Benedict: Yes; nine months previously he was seen in our gastrointestinal clinic. Cancer should have been seriously considered, although the ulcer was not very large.

Dr. McKittrick: I do not care how large it is, Dr. Benedict. I think there is only one way to treat ulcers of the stomach and that is to treat them all in the hospital as soon as the diagnosis is made. The patient should become symptom free and the ulcer heal and remain healed, or else operation is indicated.

Dr. Benedict: I agree, but the patient would not come in.

Clinical Diagnosis

Peptic ulcer, benign.

Dr. Pittman's Diagnosis

Recurrent gastric ulcer.

Anatomical Diagnosis

Carcinoma of the stomach, with secondary peptic ulceration.

Pathological Discussion

Dr. Mallory: This patient was operated on, and a lesion was found, which could not be differentiated from a benign ulcer. It was resected, and in gross it was impossible for the pathologist to say that it was anything other than a benign ulcer. When microscopic sections were made, it was apparent that there was something more. There was a deep area of peptic ulceration, lined by a typical fibrinoid layer, that had completely penetrated the muscularis and produced extensive scarring of the serosa. One small lymph node was drawn into the scar tissue, and sections through it showed a small focus, only 2 mm. in diameter, of colloid carcinoma, obviously a metastasis. Most of the ulcer appeared perfectly benign; no tumor could be found underneath the penetrating portion, but when its margins were examined it was quite clear that the mucosa, although of normal thickness, consisted entirely of malignant cells, a few of which were of the signet-ring type found in the lymph node. In most sections the tumor was entirely limited to the mucosa, but in one a submucosal lymphatic was found plugged with a thrombus of tumor cells.

Unquestionably in the interval during which the patient was observed this tumor changed from a carcinoma in situ to a frank invasive tumor, and metastasis developed. If the patient had been operated on while the cancer was still in situ, his chance of cure would have been better than 80 per cent, perhaps 100 per cent. It is doubtful if a tumor can metastasize until it begins to invade. With frank invasion and metastasis to a regional node, the chance of survival drops to about 30 per cent, even though it is still an early cancer.

CASE 27272

Presentation of Case

A twenty-two-year-old college girl entered the hospital complaining of epigastric pain and tenderness.

Five months before admission, at a time when the patient had been working very hard during and after school, she suddenly felt nauseated and vomited without apparent cause. The next day she began to have attacks of epigastric pain of unstated duration, which were severe enough to double her up. After several weeks of suffering, the patient consulted her physician, who made a diagnosis of peptic ulcer and prescribed hourly feedings and soda. This treatment gave only partial relief, and she found that warm milk was the most effective antidote. At no time had there been a change in bowel habits, melena, jaundice, vomiting or fever.

Two months before admission the patient married, and the attacks of epigastric pain grew worse, occurring in greatest severity about 1 a.m. At this time also the patient stated that she had had an attack of bronchitis, with fever, but no cough or hemoptysis. Six weeks before entry, she developed a sore mouth, and little blisters appeared on her gums and buccal mucous membranes. This condition grew worse until the patient was unable to eat and subsisted wholly on liquids for a few days. Her physician found that her gums were swollen and bled easily and prescribed a perborate mouth wash, orange juice, thiamine chloride and nicotinic acid, with the result that the condition cleared in five days. Three weeks before admission the epigastric pain became still severer and it was found that her temperature was 101°F.; the red-cell count was 3,200,000, the hemoglobin 50 per cent, and the white-cell count 16,000. Iron and liver extract were given, and a few days later the patient developed diarrhea, with six bowel movements daily, but no blood was noticed. Profuse night sweats appeared, and it was found that her temperature ranged from 99 to 102°F. She had missed her last two menstrual periods and had lost 12 pounds since the beginning of her illness.

The patient had had the usual childhood diseases, scarlet fever, and malaria at the age of eight years while in the tropics. The family history was irrelevant.

On examination the patient was well developed, but poorly nourished, pale and very ill. Examination of the heart, lungs and abdomen was negative except for slight epigastric tenderness; the blood pressure was 120 systolic, 48 diastolic. Pelvic examination was negative.

The temperature was 102°F., the pulse 90, and the respirations 30.

The urine was normal. The blood showed a red-cell count of 2,010,000 with a hemoglobin of 3 gm. (photoelectric-cell technic), and a white-cell count of 24,100 with 77 per cent polymorphonuclears. The sedimentation rate was 0.6 mm. per minute. The nonprotein nitrogen of the blood serum was 20 mg., and the protein 5.5 gm. per 100 cc. The blood Hinton reaction was negative. The stools were formed and black, and gave a ++++ guaiac test.

An x-ray film of the chest showed a primary complex in the right lower-lung field, with increased hilar calcification, but the lung fields were clear and the heart was not remarkable.

A gastrointestinal series showed a large mass occupying the greater portion of the body of the stomach, starting at the cardia and extending to an area approximately 4 cm. proximal to the pylorus. A large ulceration was present within the mass. The hourly follow-up films showed normal speed of transport through the small intestine. The loops of ileum were slightly dilated, and the barium within them had a somewhat pasty appearance.

One week after admission, gastroscopy was performed, and just inside the cardiac orifice about 2 or 3 cm. below the cardia was a smooth, rounded neoplasm projecting from the lesser curvature and involving the anterior and posterior walls, elevated above the surrounding mucosa 3 cm. or more and extending down at least to the angulus. In the central portion of the tumor there was an ulcerated, whitish sloughing area. In various portions there was definite hemorrhage, and old blood was present on the greater curvature. The angulus could not be identified, and the antrum and pylorus were not visible.

Three days later, peritoneoscopy was performed. A few ounces of clear fluid were aspirated from the peritoneal cavity, the liver appeared normal, the lesser curvature of the stomach lay under the

liver but was not adherent to it, and no tumor could be seen in the stomach. There was no evidence of metastatic disease throughout the peritoneum. A mass the size of an orange in the right side of the pelvis had the appearance of a benign ovarian cyst. No tumor cells were identified in the peritoneal fluid on pathological examination. Within the next two weeks the patient was given six 500-cc. blood transfusions. Her temperature varied between 100 and 105°F. for a short while, but gradually fell to 99°F.; the white-cell count fell to 10,000, and the red-cell count rose to 3,900,000.

On the fourteenth hospital day an operation was performed.

Differential Diagnosis

Dr. Thomas J. Anglem: May we see the x-ray films?

Dr. Aubrey O. Hampton: Here is the large ulcer described in the stomach, a huge ulcerating lesion involving the whole of the lesser curvature except a small portion of the antrum. This is, I assume, the mass described in the right side of the pelvis. You see it outlined here by pressure on the small bowel. The chest was normal as described.

Dr. Anglem: What is the relation of the mass to the markings here?

Dr. Hampton: The mucosal folds of the stomach are very thick all around it. The spleen and the liver are not enlarged.

Dr. Anglem: We are presented in this case with a large mass of clear-cut and definite objective data that leave very little doubt concerning the basic nature of the underlying lesions. The problem is chiefly one of filling in the details and deciding on the precise nature of the neoplasm, rather than on the fundamental nature of the disease. That seems to have been settled for us by the gastroscopist, who reported a definite neoplasm in the stomach, described as being a large, round, smooth tumor mass with a grayish sloughing ulcerated center, so that I think we may start with the assumption that we are dealing with a neoplasm. For the sake of completing the differential diagnosis, one might mention briefly in passing the possibility of granulomatous disease. The case history and the x-ray findings and the finding of the gastroscopist, however, do not seem to be consistent with a diagnosis of tuberculosis or syphilis of the stomach, and in both these diseases, which are relatively rare, the typical picture is one of rather shallow serpiginous ulceration, and does not seem to fit this picture at all. Hence I think we can dismiss granulomatous disease.

If it is neoplasm, what kind is it — malignant or benign? If malignant, is it an epithelial tumor or a tumor of mesodermal origin? On a purely statistical basis, carcinoma of the stomach is, of course, by a wide margin the commonest neoplasm of the stomach, and the story presented in this case is entirely consistent with a diagnosis of carcinoma of the stomach. But there are certain features of the story that are atypical. If it is carcinoma, it would have to be of the polypoid type to be reconciled with the finding on gastroscopy. Against the diagnosis of carcinoma is the age of the patient. Carcinoma is very uncommon at the age of twenty-two, but it cannot be ruled out entirely on that basis. Also against the diagnosis of carcinoma are the relative absence of impairment of gastric motility and emptying and the abrupt onset of symptoms. The other tumors of the stomach that must be considered are the benign and malignant tumors of mesodermal origin. These are leiomyoma and its malignant variant, leiomyosarcoma, neurogenic sarcoma and lymphosarcoma. I wonder if I can find out more about the details of gastroscopic examination before going on with the differential diagnosis. Hemorrhagic areas are mentioned. Were they in the ulcerated portion or in the surrounding, smooth portion of the tumor?

Dr. Edward B. Benedict: They were in the ulcerated portion.

Dr. Anglem: I think the least likely of the mesodermal tumors is lymphosarcoma. It is a relatively rare disease. It occurs in a form that originates in the stomach, and in a form with gastric involvement arising on the basis of a generalized lymphosarcomatosis. The picture presented on gastroscopy in this case, however, does not seem to be consistent with lymphosarcoma. We have here a large round, smooth, hard mass with an ulcerated center. Lymphosarcoma usually presents smaller masses than the one described here, although occasionally a large lobulated irregular polypoid tumor does occur. Some of the features of gastric lymphosarcoma are a soft consistence, with marked general vascularity and with scattered hemorrhagic areas in various parts of the tumor. I do not gather from the description of the findings on gastroscopy that they were consistent with this diagnosis.

Of the other mesodermal tumors that occur in the stomach, one stands out as the most likely diagnosis in this case, the malignant variant of leiomyoma, leiomyosarcoma. The history is entirely consistent with this diagnosis. There are certain features of the history that are difficult to reconcile, but in general it fits perfectly adequately.

Leiomyoma is the commonest benign tumor occurring in the stomach, and its malignant variant, although much less common than carcinoma, is not rare. It characteristically manifests itself by causing gastric distress or pain, and is associated frequently with epigastric tenderness of varying degree. A very common symptom is hemorrhage, causing either hematemesis or melena, or both. That is one of the features in this case which is difficult to reconcile with the picture presented by the patient on admission. We are told that the patient had a ++++ guaiac test on the stools at admission, and that the stools were dark, but beyond that there is no previous mention of either hematemesis or melena. Otherwise she presented a story that is entirely consistent with the clinical features presented usually by people with this disease: epigastric distress, starting abruptly in a patient previously in apparently good health, followed by increasing epigastric pain and clinical evidence in the patient's general decline, suggesting chronic blood loss in spite of the absence of clear-cut hematemesis or melena. The gastroscopic findings are entirely typical, presenting the usual picture, seen in leiomyoma or leiomyosarcoma of the stomach, of a round smooth mass occupying a large portion of the stomach wall, either anterior or posterior, with a central ulcerative area, frequently associated with bleeding points or hemorrhage from that area. Also characteristic of this disease is the relative absence of impairment of gastric function. There is very little, if any, impairment of gastric emptying, which we should hardly expect if the patient had a cancer of this size.

The next most likely possibility of the connective-tissue tumors that must be considered is neurogenic sarcoma. Neurogenic sarcoma can simulate leiomyoma and leiomyosarcoma in every detail, so that it is impossible to make a differential diagnosis between them on clinical grounds. The diagnosis is usually made by the pathologist.

There are certain features of this case that seem difficult to relate to the diagnosis that I have suggested, for example, the high fever and the very marked degree of anemia without a definite clinical history of blood loss by hematemesis or melena, but I believe that the answer to this apparent discrepancy is that this patient did have severe chronic blood loss in spite of the absence of such a history. I believe that the high fever and the anemia are the result of severe recurrent hemorrhage into the gastrointestinal tract. In support of that opinion is the finding that the patient responded very quickly and very readily to repeated transfusion, with a rapid rise in red-cell count and a rapid fall in fever. There are minor symptoms, which I shall mention in passing, that I do not believe have any relation to the fundamental underlying disease. The stomatitis mentioned may have developed on the basis of vitamin deficiency, or may have been due to dehydration or faulty oral hygiene. The fact that it responded quickly to vitamin therapy suggests that it may have been due to vitamin deficiency. The amenorrhea may have been the result of chronic blood loss or possibly of pregnancy. The findings on peritoneoscopy I do not believe help us much, except to exclude a generalized malignant lesion. I am willing to accept the gastroscopist's opinion that the pelvic mass was an ovarian cyst. I believe this patient most probably had a leiomyosarcoma of the stomach, with hemorrhage into the gastrointestinal tract, with neurogenic sarcoma as the second choice, and benign cyst of the ovary.

DR. TRACY B. MALLORY: Dr. Hampton, do you want to express an opinion on the basis of the x-ray findings?

DR. HAMPTON: I think Dr. Anglem has the wrong impression from Dr. Benedict's story. I do not consider it fair to say that the tumor mass was round.

DR. BENEDICT: Do not forget that I won a dollar from the X-ray Department on this case.

DR. HAMPTON: I do not care if you did. I should say that the mass was relatively flat, with a rolled margin. The whole mass was not round, and the area of ulceration is very extensive and not very deep. That is more like lymphoma than leiomyosarcoma.

DR. BENEDICT: The edges of it certainly were rounded.

DR. HAMPTON: Yes; the edges were, but the mass was not.

DR. MALLORY: Have you anything further, Dr. Benedict?

DR. BENEDICT: No; I think Dr. Anglem is to be congratulated on his discussion. He has considered everything. I was unable to reach a positive diagnosis. I mentioned the most likely things, —carcinoma, leiomyosarcoma and lymphoma,— but was not willing to say which it was. We considered an open-tube gastroscopy with biopsy to establish a positive diagnosis, but decided that the patient should be explored anyway, so that we did not do it.

CLINICAL DIAGNOSES

Carcinoma of stomach?

Dr. Anglem's Diagnoses

Leiomyosarcoma of stomach?
Neurogenic sarcoma?
Ovarian cyst, benign.

Anatomical Diagnosis

Fibrosarcoma of stomach.

Pathological Discussion

Dr. Mallory: This patient was operated on, and a subtotal gastrectomy was done. A large tumor involved the lesser curvature almost from the cardia to the pylorus, and was extensively ulcerated. It had involved the entire thickness of the gastric wall and had reached the serosa. In gross it was no more possible to make an exact diagnosis than it was on clinical examination, but microscopic examination showed a rapidly growing spindle-cell sarcoma that looked more like fibrosarcoma than leiomyosarcoma. A few mitotic figures and a great many tumor giant cells were present. However, no visible metastases were found. A great many enlarged nodes were present in the attached omentum, but they showed only inflammatory hyperplasia and one would not expect this tumor to go to the nodes in any case. I should think the picture on the whole bore out Dr. Hampton's contention as to the appearance of the tumor. The margin was round, but the tumor on the whole was rather flat, with a very extensive irregular area of shallow ulceration.

Dr. Hampton: Is that more typical in gross of lymphoma than leiomyosarcoma?

Dr. Mallory: Yes. A diffuse spindle-cell sarcoma of the stomach, with widespread invasion of the stomach wall, is quite uncommon. I remember one other very much like this, but ordinarily a spindle-cell tumor is quite well localized and has a tendency to be spherical.

Dr. Hampton: The ulcer is usually much smaller and much deeper. I think that is the reason we lost a dollar. Next time we shall win it.

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The New England
Journal of Medicine
Formerly
The Boston Medical and Surgical Journal
Established in 1828

Owned by The Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

Official Organ of
The Massachusetts Medical Society
and
The New Hampshire Medical Society

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Subscription Terms: $6.00 per year in advance, postage paid, for the United States; Canada, $7.04 per year, Boston funds; $8.52 per year for all foreign countries belonging to the Postal Union.

Material for early publication should be received not later than noon on Saturday.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston, Massachusetts.

NURSING LEGISLATION

In 1941, a bill to put control of nurses' registration and of nurses' training schools in a board of five nurses, with no representatives of the hospitals, physicians or public, was introduced by the Massachusetts Nurses Association. Hearings were held before the Committee on State Administration, and this committee has now reported out its own bill (House 2530), which increases the Board of Registration to four nurses and three physicians, including the secretary of the Board of Registration in Medicine *ex officio*. It provides for a paid nurse assistant to the secretary. This board is given control of registration.

The committee's bill also creates an approving authority to control all matters pertaining to training schools. This authority was composed as follows: a nurse who is a faculty member of an approved school for nurses and a member of the Board of Registration; a nurse who is a faculty member of an approved school for attendants and a member of the Board of Registration; the secretary of the Board of Registration in Medicine; the commissioner of education; the commissioner of public health; a physician who is a superintendent or assistant superintendent of a hospital having an approved school for nurses or attendants; and a physician who is a member, with professorial rank, of the faculty of an approved medical school. The Massachusetts Hospital Association asked that the trustee of a charitable hospital be added, to represent the large interest of these institutions, and the Massachusetts Medical Society requested the addition of a practicing physician. With these additions, the bill has the approval of the Massachusetts Hospital Association and the Massachusetts Medical Society.

The bill also provides for the registration of attendants and approval of their schools.

The control of nursing schools, which are generally operated by hospitals, is thus placed in a committee composed of members representing the medical profession, the hospitals, the general public and nurses, and the bill also promotes the training of nursing attendants with recognized standing, to alleviate the shortage that is already occurring in the present emergency. This is as it should be. It is most important that the committee's recommendation be enacted into law.

SIR D'ARCY POWER

The death of Sir D'Arcy Power* in England on May 18, 1941, at the age of eighty-five, brings to an end a useful career in both surgery and medical history. For nearly fifty years, D'Arcy Power was connected with St. Bartholomew's Hospital in London; he entered, as a student, in 1878 and left, as a consulting surgeon and one of the governors, in 1920. Few men have served a hospital for so long a period, and rarely has any-

*Books, pamphlets and letters by Sir D'Arcy Power are now on exhibit in the rotunda of the Boston Medical Library.

one been more faithful to his parent institution. A few years after his retirement, he wrote *A Short History of St. Bartholomew's Hospital, 1123–1923* (1923), a fine example of what a hospital history should be. The chapters are brief and lively, and the illustrations, one in color showing the tomb of Rahere, founder of the hospital, form a unique feature of this admirable book.

The history of his own hospital, however, was not, by any means, the chief writing of D'Arcy Power. His bibliography goes back to 1881, when he published a little book, *Manual for the Physiological Laboratory*, with Vincent Harris, the demonstrator of physiology. At that time he was assistant demonstrator of the St. Bartholomew's Hospital Medical School and had joined the hospital staff in a subordinate position only a few years before.

As time went on, he continued to publish numerous works. One of his early books was *Surgical Diseases of Children* (1895), and in 1908 he edited *A System of Syphilis*, in six volumes, containing, among other chapters, an outstanding "Introduction" by Jonathan Hutchinson and a "History" by Iwan Bloch. These two sections gave the books a distinctive character, and along with the other contributions, all of them authoritative, made this system one of the great medical publications of all time. The work, moreover, may be read today with profit, and the "Introduction" and "History" are permanent contributions to literature.

D'Arcy Power also revised *Plarr's Lives of the Fellows of the Royal College of Surgeons of England* (1930), which consisted of two volumes. This work entailed a tremendous amount of careful research and is a monumental achievement. In historical fields, moreover, Power edited a number of texts, such as John Arderne's two books, *Treatises of Fistula in Ano* (1910) and *De Arte Phisicali et de Cirurgia* (1922).

In 1930, he came to the United States as visiting lecturer to the Institute of the History of Medicine of the Johns Hopkins University. His lectures were published in 1931 as *The Foundations of Medical History*, a small, but finely evaluated document, particularly relating to the early history of medicine in England, a field in which he was a well-known authority. In 1931, his *Selected Writings* were issued, covering the period from 1877 to 1930. Finally, in 1939, a small book, entitled *A Mirror for Surgeons: Selected readings in surgery*, was published both in Great Britain and in this country.

D'Arcy Power was a man of great charm. He made friends readily. For years he was known to the students at St. Bartholomew's Hospital as "Sunny Jim." Although he was always cheery and never complaining, life dealt him many tragic blows. His only daughter died of whooping cough at the age of two, and his younger son was killed in World War I. During his last years he was saddened by the death of his wife. He kept up his activities, however, almost to the very end, occupying his house on Chandos Street, long a mecca for friends of medical history, throughout the bombing of London and up until the beginning of the present year. He then found it no longer safe to live there, or to keep his books, of which he had a great many, in such an exposed position. One of his last letters, stating that his books would be sold at Sotheby's auction room in London, was published in the May 17, 1941, issue of the *Journal of the American Medical Association*. A catalog of these books, recently received, shows that he had collected over the years a considerable number of important medical texts, some of which will find their way to this country.

Even in his later years, D'Arcy Power was an active person. He wrote, for example, many of the obituary notices of medical men in *The Times*, of London. This meant that he had to have the material readily at hand, for the notices were prepared in the course of a few hours. For the average man, this would have been impossible, but for the quick, surgically trained Power, nothing was too much of a task, and he turned out extraordinary obituary notes for the daily press, listing not only the important contributions that the deceased colleague had made to medicine, but also giving an estimate of the man's

character and the passing or enduring value of his work.

He kept up his cheerfulness to the end of his life. In a letter written last fall from London, before he had moved into the country, he noted in a postscript: "London has become very noisy. Fortunately, I have one deaf ear and I am not, therefore, much disturbed at night." For a man in his eighty-fifth year, living in the heart of London during the height of the blitzkrieg, to write in such a fashion characterizes this forthright gentleman. Surgeon, historian, biographer, lover of mankind, keen student of human nature, delightful correspondent and charming friend, D'Arcy Power leaves behind him many memories that will long be cherished by his friends, as well as a score of monumental contributions to medical literature. It was, indeed, characteristic of him that just before he died, he turned over to the Royal College of Surgeons a supplement to *Plarr's Lives*, bringing the necrologies up to 1940.

A fitting eulogy is contained in the final paragraph of his death notice in the May 31, 1941, issue of the *Lancet*.

> And now that he has gone to his rest, he stands out not so much as a single individual, but as one of a chain of stouthearted English doctors continuing to serve mankind. For his father, coming from an old Yorkshire family, was Henry Power, the ophthalmic surgeon, a fine figure of a man. His son, D'Arcy II, is high up in the medical service of the Royal Air Force, and his grandson, D'Arcy III, is following the same profession. Here indeed is a family that have served their country well.

MEDICAL EPONYM

Hunter's Glossitis

The strongly individualistic contributions of Dr. William Hunter (1861–), pathologist to the Charing-Cross Hospital, to knowledge of the nature and causes of pernicious anemia include numerous descriptions of the glossitis that is often identified by his name. The following quotation is from his article, "Further Observations on Pernicious Anaemia (Severe Cases): A chronic infective disease: Its relation to infection from the mouth and stomach: Suggested serum treatment," which appeared in the *Lancet* (**1**: 221–224, 296–299, 371–377, 1900):

> . . . I was struck by the curious character of the sores on the tongue — localised inflamed patches sometimes showing vesicles filled with clear serum situated under the tip of the tongue, the inflamed areas shifting from time to time, with atrophic appearance of the intervening mucosa. The condition thus described is not one of ordinary stomatitis or glossitis such as one meets with as the result of the local irritation of decayed or irregular teeth. . . . Another feature I have had to note is what I may term the "periodicity" of the stomatitis — its variability from time to time, independently apparently of treatment, notably its greater severity at the outset of the disease, usually tending to subside or at least to give less discomfort as the disease advances.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

Raymond S. Titus, M.D., *Secretary*
330 Dartmouth Street
Boston

Cesarean Section, Followed by Fatal Surgical Shock

A thirty-six-year-old primipara at term, because of fetal distress, was delivered by cesarean section when the cervix was almost fully dilated.

The prenatal care had been excellent. The patient had been under observation since the beginning of the third month, during which time the blood pressure was normal and the urine showed no albumin. At about four and a half months a large intramural fibroid was removed. This did not interrupt the pregnancy. The convalescence from this operation was stormy, and during the rest of the pregnancy the patient suffered from a secondary anemia, which was treated with ferrous sulfate and liver. The patient was in the hospital for observation several times during the last two months of her pregnancy.

Three weeks after the expected date of confinement, labor started, being induced by castor oil. Because she was rated as a bad surgical risk and because it was known that the baby was small, a test of labor was considered a conservative method of handling this particular patient. Two donors were in constant attendance during the labor, as was the physician. The labor was normal. At the end of nine hours, the cervix was practically fully dilated, with unruptured membranes. At this time, the membranes were ruptured and the head settled into the pelvis. Unfortunately, however, the

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

fetal heart became irregular and the rate slowed to about 90 beats per minute. Cesarean section was decided on, because of the irregular fetal heart and the patient's age. At operation, the cord was found to be tight about one shoulder, a condition that was probably the cause of the fetal distress. The amount of blood lost at operation was said to have been little. During operation, however, the patient's blood pressure could not be obtained and the pulse was very rapid. Intravenous glucose and 500 cc. of blood were given at the end of the operation. The patient responded well but an hour later went into collapse and died.

Comment. Intramural fibroids that are removed during pregnancy may so injure the uterine wall that rupture during labor must be considered a possibility. Because of this, the patient was under close observation during labor, so that cesarean section could have been performed immediately, if there were any evidence of rupture.

When a patient is practically fully dilated and the fetal heart shows distress, it is always questionable whether cesarean section is a justifiable procedure. One would hesitate, knowing that the wall of the uterus had been weakened by a myomectomy, to perform a version. With full dilatation and the head in mid-position, forceps in the hands of a skilled operator would probably have guaranteed a live baby about as well as laparotomy.

Shock is a peculiar thing. This patient apparently did not die of hemorrhage, but the laparotomy was more than she could stand. Everything was done that could have been done, — the patient was transfused, after which she rallied, — but in spite of this, she sank into collapse and died.

COMMITTEE ON PUBLIC RELATIONS

It seems likely that many members of the Society would be interested in the following resolutions, which were recently passed by the medical staff of the Cape Cod Hospital and approved by its executive committee.

ELMER S. BAGNALL, *Secretary*

* * *

WHEREAS, If we are not at war, we are in a state of grave emergency; and

WHEREAS, Both federal and state governments wish the regular forces strengthened by volunteers; and

WHEREAS, The enrollment of volunteers is hampered, first, by fear of personal injury and, second, by lack of measures to see that volunteers will be cared for, if injured in training or action; be it therefore

RESOLVED, That the Cape Cod Hospital and Cape Cod Hospital staff mutually agree to assure free hospitalization and medical care for any such volunteers to the limit of their resources, volunteers to be certified to have been injured in line of duty, while in training or on public service, and to include the State Guard; and be it further

RESOLVED, That should future plans or laws provide payment for such care and should payment be made for such care, the Cape Cod Hospital staff agrees to add such sums to the endowment fund of the Cape Cod Hospital.

DEATHS

ANDERSON — BERTHA O. ANDERSON, M.D., of Pittsfield, died June 25. She was in her fifty-ninth year.

Born at Lawrence, Kansas, she received her degree from the University of Kansas School of Medicine in 1911. From 1911 to 1929 Dr. Anderson practiced general medicine, surgery and obstetrics in Lincoln, Illinois. From 1931 to 1935 she practiced in Lakewood, Ohio, specializing in diseases of the gastrointestinal tract, gynecology and proctology. She had taken postgraduate work at the New York Polyclinic Hospital and Harvard Summer School, and had done observation work at the Mayo Clinic in Rochester, Minnesota. She moved to Pittsfield in 1935 because of ill health.

Dr. Anderson was a member of the Massachusetts Medical Society and the American Medical Association. She was also a member of the National Women's Medical Association, the Women's Medical Association and the Lakewood (Ohio) Medical Club.

A brother, a nephew and a niece survive her.

BURNETT — FRANK H. BURNETT, M.D., of Brockton, died June 21. He was in his seventy-sixth year.

Born in Guilford, Vermont, he attended Dartmouth College and received his degree from Dartmouth Medical School in 1890. Dr. Burnett was on the staff of the Brockton Hospital and was a former president of the Plymouth County Medical Society. He was a member of the Massachusetts Medical Society and the American Medical Association.

His brother survives him.

McCROSSAN — CHARLES L. McCROSSAN, M.D., of Somerville, died June 26. He was in his fifty-sixth year.

Born in Somerville, he attended Harvard University and received his degree from Harvard Medical School in 1910. At the time of his death he was a member of the Somerville Board of Health and was on the staff of the Somerville Hospital. He was a member of the Massachusetts Medical Society, the American Medical Association and the New England Obstetrical and Gynecological Society.

His widow, his father, a daughter, a son and two brothers survive him.

MULHOLLAND — BERNARD J. MULHOLLAND, M.D., of Lawrence, died April 16. He was in his fifty-ninth year.

Born in Lawrence, he received his degree from Baltimore Medical College in 1906, and started private practice in Lawrence.

He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow and two daughters survive him.

VICKERY — EUGENE A. VICKERY, M.D., of Wellesley, died June 22. He was in his sixty-third year.

Dr. Vickery received his degree from Harvard Medical School in 1903. He was a fellow of the Massachusetts Medical Society and the American Medical Association. At the time of his death he was Commander, Medical Corps, United States Navy (retired). He was a member of the Southeastern Surgical Congress and a fellow of the American College of Surgeons.

His widow survives him.

WALSH — JAMES H. WALSH, M.D., of Fall River, died May 14. He was in his fifty-eighth year.

Born in Fall River, he received his degree from the College of Physicians and Surgeons of Baltimore, Maryland, in 1910. He was a former president of the Fall River Medical Society, and was a member of the Advisory Board of the Union Hospital. For thirty years he was bacteriologist for the City of Fall River and was associate medical examiner for several years. For many years he was pathologist at the Union, St. Anne's and General hospitals, of Fall River, and the Newport Hospital of Newport, Rhode Island. At the time of his death he was on the Selective Service Board.

Dr. Walsh was a fellow of the Massachusetts Medical Society and the American Medical Association.

His widow, a daughter and a son survive him.

CORRESPONDENCE

NEW REGULATIONS IN REGARD TO COMMUNICABLE DISEASES

To the Editor: In 1938 an amendment to Section 6 of Chapter III of the General Laws authorized the Department of Public Health to make rules and regulations in regard to the control of communicable diseases. Such regulations were formulated and approved on August 9 of that year and were printed in the September 15, 1938, issue of the *Journal*.

In October of the same year, "infectious encephalitis" was made reportable, replacing the old terminology of "encephalitis lethargica." A note in regard to this change appeared in the November 10 issue of the *Journal*.

On May 13, 1941, further additions were made to the list of reportable diseases, and certain amendments were made in the isolation and quarantine requirements to bring them in line with the latest revision of the recommendations of the Subcommittee on Communicable Disease Control of the American Public Health Association. These changes are given below.

Printed copies of the revised regulations will be available for distribution in the very near future. The revised list of reportable diseases is also being reprinted and will be sent to every physician in the State.

I should also like to call attention to the fact that the rules and regulations for the treatment of persons exposed to rabies were amended so that the first sentence of Rule 2 reads as follows: "Antirabic vaccine and antirabic treatment shall be furnished by the board of health for all persons bitten on the head, or in cases with severe multiple lacerations." The amendment consists of the addition of the last phrase of this sentence.

PAUL J. JAKMAUH, M.D.
Commissioner of Public Health

State House
Boston

CHANGES IN ISOLATION AND QUARANTINE REQUIREMENTS

List of Reportable Diseases

Lymphocytic choriomeningitis and Weil's disease were added to the reportable list, and all forms of meningitis were made reportable. "Paratyphoid fever A and B" was changed to "Paratyphoid fever and all other Salmonella infections."

Periods of Isolation and Quarantine

Chicken pox. (Column 2.) Amended to read as follows: "One week from appearance of eruption and thereafter until crusts have disappeared, provided that total period of isolation shall not exceed fourteen days."

Cholecystitis of typhoid origin. (Columns 4 and 5.) Omitted "No restrictions" and substituted "Same as typhoid fever."

Diphtheria. (Column 4.) Omitted "or on the basis of a previous attack of the disease."

Dysentery, amebic. (Column 2.) Omitted "Same as typhoid fever" and substituted "No restrictions except for foodhandlers, who shall be kept from their occupations until three successive negative stool examinations, secured at intervals of not less than three days apart, shall have been obtained." (Column 3.) Omitted "Note 3" and substituted "No restrictions except for foodhandlers, for whom restrictions are same as for case."

Dysentery, bacillary. (Columns 4 and 5.) Omitted "No restrictions" and substituted "Same as typhoid fever."

Infectious encephalitis. (Column 1.) Order of words changed to "Encephalitis, infectious," and placed in table immediately after "Dysentery, bacillary."

Lobar pneumonia. (Column 1.) Order of words changed to "Pneumonia, lobar," and placed in table immediately after "Plague."

Lymphocytic choriomeningitis. (Column 1.) Placed in table immediately after "Leprosy." (Column 2.) "Until recovery." (Columns 3, 4 and 5.) "No restrictions." (Column 6.) "No."

Measles. (Column 5.) Changed "Note 7" to read "Note 8."

Meningococcus meningitis. (Column 1.) Order of words changed to "Meningitis, meningococcal."

Ophthalmia neonatorum. (Column 2.) Amended to read: "One week after subsidence of symptoms. In gonococcal ophthalmia, thereafter until two successive smears from each eye at an interval of not less than forty-eight hours are negative for gonococci."

Paratyphoid fever. (Column 1.) Omitted "A and B" and substituted "and all other Salmonella infections." (Columns 4 and 5.) Omitted "No restrictions" and substituted "Same as typhoid fever."

Pfeiffer bacillus meningitis. (Column 1.) Amended to read: "Meningitis, other forms, such as Pfeiffer bacillus, pneumococcal, streptococcal, etc." and placed in table immediately after "Meningitis, meningococcal." (Column 2.) Omitted "No restrictions" and substituted "Until recovery."

Tuberculosis. (Column 3.) Omitted "No restrictions" substituted "Note 7."

Typhoid fever. (Columns 4 and 5.) Omitted "No restrictions" and substituted "No restrictions provided contacts can be relied upon to observe precautions outlined by board of health and provided at least one satisfactory stool specimen is submitted for examination."

Weil's disease, infectious jaundice due to *Leptospira icterohemorrhagiae.* (Column 1.) Placed in table imme-

diately after "Undulant fever." (Column 2.) "Until recovery." (Columns 3, 4 and 5.) "No restrictions." (Column 6.) "No."

Notes

Note 3. In line 1 substituted "household" for "family." Omitted the words "amebic or" and substituted the word "fever" for the phrase "A or B." Added the following sentence to the note: "Foodhandlers living in a household with a recovered case which continues to excrete typhoid bacilli after convalescence shall be excluded from their occupations unless they have been inoculated with typhoid vaccine within two years."

Note 6. Amended to read: "Patients with open tuberculosis should in most cases receive sanatorium treatment, both for the benefit of the individual and the protection of his family. Those who remain in their homes shall observe all precautions necessary to prevent infection of the members of their families and of others with whom they may come in contact. This shall include approved methods of collection and disposal of the sputum, the sterilization of any articles of clothing and of toilet articles which may become contaminated by the sputum, the use of separate dishes and eating utensils and proper sterilization of the same. The patient should sleep in a separate room. For details concerning precautions in home care a pamphlet of the Massachusetts Department of Public Health entitled *Home Care of Tuberculosis Patients* is available.

"As soon as a diagnosis of tuberculosis has been established, arrangements should be made for the examination, including an x-ray of the chest, of all members of the immediate family and of other persons with whom the patient has been in close contact. If the family cannot afford x-ray examination by a private physician, facilities are available through the various state, county and municipal sanatoria. Persons with suspicious findings and those who have had contact with a tuberculous patient should be kept under medical observation as long as advised by the physician. It is the responsibility of the local board of health to provide hospital care for cases of tuberculosis, when needed, and to see that contacts are examined where such examinations have not been made through a private physician.

"When a case is reported the public health nurse representing the board of health should visit the patient's home. She should instruct the family in the sanitary precautions described above, see that arrangements are made for the examination of contacts and if necessary provide transportation to the place where they are to be x-rayed, and should aid the patient in obtaining admission to a sanatorium if this has been recommended by his physician.

"Thereafter, the nurse should make visits to the home at least once in six months to determine whether the patient has moved, whether the above-mentioned precautions are still being observed, and whether any new measures are needed to control the spread of the disease. If the patient has moved to another town or state, the Massachusetts Department of Public Health should be notified. In cases where the physician wishes to exercise complete supervision, the nurse should obtain such information from him.

"The only acceptable reasons for the board of health failing to exercise the supervision outlined above are: refusal of the family physician to permit periodic visits by the nurse; or placing of the patient's name on an 'inactive list' as a result of examination, including x-ray.

"No person who has or who has had tubercle bacilli in the sputum or other bodily discharges shall be allowed to engage in teaching, nursing, dairying or occupations involving food handling or the care of children until he has received a certificate from the board of health stating that his employment would not be dangerous to the public health."

Note 7. Substituted "Persons living in a family in which a case of tuberculosis exists or has existed within two years, and whose occupations involve food handling or contact with children, shall be required to have an x-ray of the chest to determine whether they shall be allowed to continue in such occupations."

Note 8. Former "Note 7," renumbered.

ARTICLES ACCEPTED BY THE COUNCIL ON PHARMACY AND CHEMISTRY, AMERICAN MEDICAL ASSOCIATION

To the Editor: The following products have been accepted by the Council on Pharmacy and Chemistry since May 1, 1941:

Armour Laboratories

Suprarenalin Solution 1:1000, 5-cc. vial (for hypodermic use)

Geo. A. Breon & Co., Inc.

Tablets, Ascorbic Acid — Breon, 25 mg.
Tablets, Ascorbic Acid — Breon, 100 mg.

Cutter Laboratories, Inc.

Sobisminol Solution — Cutter, 50-cc. bottle

Drug Products Co., Inc.

Pulvoids, Thiamine Hydrochloride, 1 mg.
Hyposols, Solution of Thiamine Hydrochloride Crystals, 6.66 mg. per cc., 1-cc. ampuls and 10-cc. and 30-cc. vials
Hyposols, Solution of Thiamine Hydrochloride Crystals, 10 mg. per cc., 1-cc. ampuls and 10-cc. and 30-cc. vials
Hyposols, Solution of Thiamine Hydrochloride Crystals, 33.33 mg. per cc., 1-cc. ampuls and 10-cc. and 30-cc. vials
Hyposols, Solution of Thiamine Hydrochloride Crystals, 50 mg. per cc., 10-cc. ampuls and 10-cc. and 30-cc. vials
Hyposols, Solution Procaine Hydrochloride 2 per cent, 2-cc. ampuls

Endo Products, Inc.

Ampoules, Epinephrine in Oil 1:500 — Endo, 1 cc.
Tablets, Nicotinic Acid — Endo, 50 mg., scored
Tablets, Nicotinic Acid — Endo, 100 mg., scored

Flint, Eaton & Co.

Tablets, Sulfanilamide, 1 gr.
Tablets, Sulfanilamide, 7.7 gr.
Tablets, Sulfapyridine, 0.5 gm. (7.7 gr.)
Sulfathiazole Tablets, 0.5 gm. (7.7 gr.)

Lederle Laboratories

Tablets, Aminophyllin — Lederle, 0.2 gm. (3 gr.)

Eli Lilly & Company

Tablets, Sulfathiazole — Lilly, 0.25 gm. (3¼ gr.)
Tablets, Sulfathiazole — Lilly, 0.5 gm. (7½ gr.)

National Drug Company

Tablets, Sulfanilamide, 1 gr.
Tablets, Sulfanilamide, 7½ gr.

Schieffelin & Co.

Sulfanilamide Tablets, 7½ gr.

Smith-Dorsey Company

Tablets, Sulfapyridine, 0.5 gm. (7.7 gr.)

Frederick Stearns & Co.

Ascorbic Acid Tablets — Stearns, 50 mg.
Ascorbic Acid Tablets — Stearns, 100 mg.

Upjohn Company

Typhoid Vaccine — Upjohn, six 2½-cc. vials, package
Tablets, Sulfapyridine — Upjohn, 0.5 gm. (7.7 gr.)
Tablets, Sulfathiazole — Upjohn, 0.5 gm. (7.7 gr.)

Winthrop Chemical Co., Inc.

Pontocaine Base Eye Ointment

OFFICE OF THE COUNCIL
By: C. C. Bean

535 North Dearborn Street
Chicago, Illinois

REPORTS OF MEETINGS

HARVARD MEDICAL SOCIETY

A regular meeting of the Harvard Medical Society, held at the Peter Bent Brigham Hospital on April 8, was inaugurated by the usual presentation of a case.

A middle-aged woman had experienced a fall eight months before admission, with the subsequent development of headache and dizziness. She had had known hypertension for two years. For two weeks prior to entry, there had been extreme vertigo, with a marked tendency to stumble to the left and with weakness of the right side. Physical examination was remarkable chiefly for moderate cardiac enlargement and hypertension (160 systolic, 100 diastolic). On neurologic appraisal, there was weakness of the right arm and leg associated with diminished tendon reflexes. Poor co-ordination of the left arm and leg was noted. Pain sense was diminished on the left trunk and leg. All laboratory data, including examination of the cerebrospinal fluid, were negative. The electroencephalogram revealed diffuse abnormalities from all areas, and a cystometrogram indicated the presence of a cord bladder.

Dr. Soma Weiss stated that this was an example of an unusual syndrome, thrombosis of the inferior cerebellar artery — in this case, on the left side. This condition results in pyramidal-tract signs on the opposite side, and cerebellar and sympathetic manifestations on the homolateral side. Dr. E. A. Stead, Jr., remarked that the disease is rare in that the symptoms and signs result from central rather than peripheral paralysis. Therefore, a dissociation of symptoms, with some normal as well as abnormal responses, may result.

The speaker of the evening was Dr. Walter F. Dearborn, of Harvard University, whose subject was "Disabilities in Reading and Their Remedying." In introduction, it was emphasized that these difficulties may occur at any level of educational life and may cause late as well as early failures. A welter of causes has been suggested, which may be environmental, educational or organic. Poor training, large classes, absence from school, faulty home facilities and so forth may contribute a share of reading disability. Organic causes, such as difficulty with articulation, deviations in eye and hand dominance and anisopia, probably function largely as initial handicaps that trip only a few students, whereas multiple structural changes are frequent. Visual acuity and eye-muscle imbalance are becoming less popular as causative factors.

Dr. Dearborn believes that laterality and mixed dominance play a role. Even opponents agree that left-eye dominance favors right-to-left eye movements. Although something may be gained by waiting in such cases, Dr. Dearborn suggests "changing the menu" in the meantime to avoid the early emotional upsets that may occur during the process of expectant waiting. Furthermore, time alone will not always bring the desired results.

A study of left ocular and manual dominance indicates that persons with such findings are retarded in about 40 per cent of cases, but that the greatest degree is noted in those with mixed dominance of hand and eye. This may cause an aversion to reading if nothing is done at an early date. This aversion may be accentuated in an intelligent person who has some other special skill, and in such cases it is particularly worth while to treat left lateral or mixed dominance.

Anisometropia, with poor fusion of the images of the two eyes, causes an extra effort and makes reading a difficult task. This was present in 50 per cent of extreme cases of disability, as against 23 per cent in a control group. It is probably a factor in certain cases, therefore, but admittedly not an insurmountable one. In many cases of reading disability, one may discover a chief cause, but just about as frequently multiple well-recognized causative factors are present.

The chief remedy is to have only one capable instructor with a unity of purpose. Too many schools merely complicate matters, and no one is able properly to treat cases of reading disability, even when the diagnosis is made. The problem is most acute in the young, but is far from absent at college age. Freshman training at Harvard University is now accomplished by means of motion pictures graded for speed. It has been found possible in this way to increase the speed and accuracy of reading and to elevate the college grades as compared with controls. More recently, stress has been laid on comprehension rather than mere reading ability.

The discussion was opened by Dr. Edwin M. Cole, of Harvard Medical School. For organic disease or injury to cause aphasia the lesion must be unilateral on the dominant side. He has been impressed with the hereditary aspects of the dominance problem and the high incidence of stuttering, left-handedness and reading disability in such families.

Dr. Virgil G. Casten stated that an ophthalmologist's sole purpose in cases of reading disability should be to rule out organic eye disease, although even such seemingly serious findings as anisoconia seldom cause such difficulty of themselves.

In conclusion, Dr. Dearborn stated that anyone with reading disability can be either taught or improved if enough time and patience are employed.

EVANS MEMORIAL LECTURE

At a Robert D. Evans Memorial Lecture at the Evans Memorial, Massachusetts Memorial Hospitals, on March 28, Dr. Allen O. Whipple, of Columbia University, discussed "Certain Splenopathies in Relation to the Vascular Bed of the Spleen." The discussion was based on 483 splenopathies observed in the Combined Spleen Clinic, of which 237 were operated on, with a mortality rate of 8 per cent.

There has been much discussion about the character of the vascular bed of the spleen, some observers considering

it an open system wherein there is no actual connection but merely a vascular bed between the arterioles and venules. The opposite view of a closed system with terminal arterioles communicating directly with the venous sinuses has been proposed, and some observers believe in a combination of the two extreme views. Between 1936 and 1938, it was considered that ample evidence was at hand to indicate that a closed system obtained under normal conditions, and that any suggestion of a vascular bed was the result of handling or improper technic. An improvement of the technical details whereby the spleen may be directly observed in its functioning physiologic state seems to contradict these previous findings, and it is now believed that this new method of refractibility may make anatomic structure and physiologic function coincide. At any rate, certain positive statements can be made: the pulp spaces are the chief, if not the only, link between the arterioles and veins; these are variable in size and shape, with interconnections; their size is such that cells as well as fluid can pass through; they may dilate to several times the size of an erythrocyte or contract to almost nothing; the spaces are the only site of circulation and storage of and contact with phagocytes, serving as a means of bringing the circulating blood in contact with the pulp-space cells for phagocytosis and destruction of erythrocytes. Only a few important conditions associated with this pulp system were discussed.

In chronic microcytic hemolytic anemia, there may be a hemolysin at work, or inferior erythrocytes, in the form of spherocytes, which are easily destroyed, may be present. This was not debated. The importance of the spleen in this condition is proved by the effect of splenectomy. Microscopically there is engorgement of the pulp spaces with spherocytes, and the venous sinuses are collapsed by the pressure of the distended spaces. Since the spherocytes are unable to pass, owing to their inflexible nature and their lack of rouleaux formation, they are exposed for longer periods to any hemolyzing factor in the pulp-space walls, and are either sensitized or directly destroyed by the hemolysin. There is evidence of an enhanced fragility and of a larger number of such cells in the arterioles and pulp spaces than in the venules. This indicates that it is in these spaces rather than in the venous sinuses that the cells are in proximity with phagocytes.

Hypertensive splenomegaly, which is considered a congestive disease, is usually signified clinically as Banti's syndrome of hepatosplenomegaly. The present concept of this condition, which also includes the finding of anemia, leukopenia and gastrointestinal hemorrhages, includes many cases not originally considered by Banti, and excludes some cases following the original description. This syndrome may be caused by any obstruction to flow within the portal vein, but only if there is a gradual increase of portal-vein pressure while the peripheral venous pressure remains essentially unchanged. The site of obstruction may be intrahepatic or extrahepatic. Sixty-eight per cent of 140 cases were directly attributable to cirrhosis of the liver. The amount of hypertension and splenomegaly is proportional to the type of cirrhosis, that due to *Schistosomum mansoni* causing the greatest degree. This can be reproduced in dogs by the injection of silica particles in the portal vein over a period of about sixteen months. Laënnec's cirrhosis or periportal fibrosis causes a variable degree of hypertension, and these patients are prone to succumb to liver insufficiency, the chief pathological findings being in the lobules rather than in the periportal areas. Among the extrahepatic causes of the Banti syndrome may be mentioned a traumatic pancreatic cyst wherein operation effected a cure. In infants and children a congenital block of thrombosis may initiate the condition. Early operation should be carried out, and a liver biopsy always performed, for only those without cirrhosis of the liver may be cured. No subsequent cirrhosis has been known to develop if there was none at operation, whereas patients with cirrhosis have recurrences of all manifestations. Histologically, these spleens have distended venous sinuses, diminished pulp spaces and fibrosis.

BOOK REVIEWS

The Head and Neck in Roentgen Diagnosis. By Henry K. Pancoast, M.D., Eugene P. Pendergrass, M.D., and J. Parsons Schaeffer, M.D., Ph.D. 4°, cloth, with 1251 illustrations. Springfield, Illinois: Charles C Thomas, 1940. $12.50.

This book is undoubtedly the best textbook of its kind in the English language. It is encyclopedic in character and is profusely illustrated with reproductions of roentgenograms, anatomic illustrations, drawings, photographs and even a few colored plates. The compilation and arrangement of such a monumental work must necessarily mean not months but years of arduous labor, and the painstaking accuracy with which the vast volume of material is presented is a tribute to the diligence and indefatigable attention of the authors. The book is a real monument to the memory of Henry K. Pancoast, the senior author, who died while it was still in an embryonic stage, and the major credit should go to the junior author, E. P. Pendergrass, who carried it to completion, with the help of the associate author, J. Parsons Schaeffer, who has added the valuable anatomic groundwork on which the roentgenographic material is based. The book covers every conceivable method, procedure and process in roentgenography, as well as the rules and precepts of diagnosis. It is important to know the methods of examination and the technical aids that can be used, as well as the normal anatomic relations of each part examined. Both features are well covered.

The subject matter is unusually wide in scope, since the authors cover not only the skull and brain, which alone would make quite a monograph, but also the sinuses, the teeth and jaws, the mastoids, the eyes, nose and lachrymal passages, and the neck — not only the cervical spine, but also the soft tissues of the neck and the cervical spinal cord. It is indeed a very comprehensive and complete book, and very accurate and reliable, and yet there are a few faults to be found, since no book is ever quite perfect. One might grade it "ninety-nine per cent," and thus take away emphasis from the one per cent of criticism. The reviewer has observed only two major faults: The anatomy, although detailed and no doubt quite accurate, might well have been correlated better with the roentgenologic viewpoint and the descriptive text. As written, the anatomic descriptions stand out like islands in the roentgenologic current. The other minor defect is the fact that quite a few of the illustrations were apparently made from old films or plates and are lacking in sharpness, contrast and detail such as everyone expects routinely today. This is perhaps unavoidable, since one cannot expect to accumulate perfect examples of the thousand and one varieties of disease in a year or two. The authors have wisely borrowed illustrations from their confreres and have been most careful to give due credit for each one used.

One should make particular note of the fact that all roentgenograms are printed as negatives, that is, in their original whites and blacks — a laudable procedure, and one that other roentgenologic textbooks would do well to emulate. Another very commendable feature is the extensive and complete index, covering seventy-two pages. The index of such a book must necessarily be one of its most important parts, and yet a proper index is all too frequently slighted. Not so in this book, although there are a few errors and omissions — for example, "glioma" refers only to the description of calcification in the gliomas and not to the classification of gliomas heading that section. One might add other minor criticisms, such as the charge that the authors' classification of brain tumors as "parasellar," "metasellar" and so forth is unclear and not useful, or that pathology and clinical correlation have been slighted for anatomy, but to do so would be to emphasize the one per cent of criticism and slight the ninety-nine per cent of commendation and approbation.

The mechanics of the book are excellent — the typography is clear, the illustrations are as good as any in American textbooks (and better than most), the paper is of very good quality, and the whole make-up is highly commendable. The publishers as well as the authors are to be congratulated. On the whole, this book is valuable, comprehensive and accurate, and should be in every roentgenologist's office — on his desk for everyday use.

De morbis artificum [Disease of Workers]. A diatribe by Bernardini Ramazzini. The Latin text of 1713 — revised, with translation and notes. By Wilmer Cave Wright. 8°, cloth, 549 pp., with 3 illustrations. Chicago: University of Chicago Press, 1940. $5.00.

One of the classic texts of medicine is the book by Ramazzini, first published in 1700, on occupational diseases. An English translation has long been needed, for the book has not been put into that language since the eighteenth century, and at that time only part of it was translated. In many cases, moreover, the early translators paraphrased the text and omitted what they thought superfluous or obscure. Now, Dr. Wright, emeritus professor of Greek at Bryn Mawr College, and well known for her fine translation of Fracastorius' *De Contagione* (1546), has made an excellent English translation from the Latin text of 1713. She has, moreover, added numerous notes, and provided a sixty-four-page introduction, giving not only the history of the man but details in regard to the various printings of the book itself. Such a book must naturally be in the historical collection of every medical library. The volume is one of a series issued on the history of medicine under the auspices of the Library of the New York Academy of Medicine. Not the least useful of the series of texts published under their auspices is this book by Ramazzini.

Doctor in Arabia. By Paul W. Harrison, M.D. 8°, cloth, 303 pp., with 9 illustrations. New York: The John Day Company, 1940. $3.00.

Harrison, a graduate of Johns Hopkins, went to Arabia as a missionary doctor after finishing his internship in Boston, and settled in Muscat, on the Persian Gulf, one of the driest, hottest and most inaccessible corners of the world. Muscat was the center of the contraband traffic in ammunitions, and Harrison found a million rifles for sale in the warehouses of that little city. It was also the port for the export of Oman dates. When Harrison set up his hospital, it is interesting to note that operation for strangulated hernia was one of the commonest.

Harrison describes his patients, the public-health problems and economic and social conditions in Oman, and the daily life of a splendid, high-minded missionary doctor, working against heavy, environmental odds. He looks forward to a time when the government of Oman will organize a health service for the whole country. This would be the only way to fight the diseases commonly met with in Dr. Harrison's hospital: malaria, Madura foot, trachoma, syphilis, gonorrhea and leprosy. The members of the medical staff should be natives, and the hospital should be a department of the government, with community support behind it. One is convinced that Dr. Harrison had made out a case and that his book points the way to the value of social medicine in the isolated corners of the world.

Manual of Physical Diagnosis, with Special Consideration of the Heart and Lungs. By Maurice Lewison, M.D., and Ellis B. Freilich, M.D. In collaboration with George C. Coe, M.D. 8°, cloth, 317 pp., with 75 illustrations. Chicago: The Year Book Publishers, Incorporated, 1941. $3.00.

The appearance of so many new textbooks of physical diagnosis during the last few years may be attributed to the natural desire of each publishing company to find and sell a text that will be accepted as standard all over the country. These books generally show little originality, and vary from one another only in the degree of elaboration and pictorial illustration with which they present the old facts of physical examination combined with a certain amount of electrocardiography, and the discussion of a few groups of clinical syndromes, particularly "diseases of the chest and circulatory organs." Some of them, such as those offered by Martini, by Buck and by Clendening, limit their subject matter to the noninstrumental examination of the patient. This book is an example of the simpler type of text, and is to be recommended on this account.

There is remarkably little fault to be found in respect to its accuracy, although the old American legend of physiologic dullness of the apex of the right lung is dutifully repeated. A defect is that bibliographic references which might aid the student to develop the habit of consulting original sources are entirely lacking.

Bacteriology in Neuropsychiatry: A survey of investigations concerned with the specific role of infectious and immune processes. By Nicholas Kopeloff, Ph.D. 8°, cloth, 316 pp., with 11 tables. Springfield, Illinois: Charles C Thomas, 1941. $4.50.

The reviewer has rarely met with a book that seems to have as slight a value to the medical profession as this one. All the material of any worth is easily found in common medical literature. The second half of the volume is padded with speculations of no apparent scientific value, and there are large sections on treatment, which seem to be wholly out of place in a book on bacteriology. The literature is widely quoted, but with little discrimination. The book, unfortunately, is finely published, and the splendid reputation of the publisher may catch the unwary.